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Air Toxics Problem in the United States An Analysis of Cancer Risks for Selected Pollutants

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The report presents estimates of the national cancer incidence associated with about 25 common potential carcinogens found in ambient air. Exposures are estimated using both air quality data and emission-dispersion model techniques.

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PREFACE

This report on the air toxics problem in the United States is a final version of a September 1984 U.S. Environmental Protection Agency draft document entitled "The Magnitude and Nature of the Air Toxics Problem in the United States." Simultaneously with release of the draft report, EPA solicited comments on the analysis from a peer review panel made up of non-EPA experts in fields such as toxicology, air monitoring, and air pollution control. In response to the panel's comments, as well as to unsolicited comments received from several organizations, the authors have substantially revised the Executive Summary and have made changes in other parts of the report. The revised version more clearly delineates the limitations and caveats of the analysis. Also, the title of the report has been revised to recognize the limited scope of the analysis.

In addition, certain of the risk estimates have been changed as new information has become available. In particular, the risk estimates for nickel and ethylene dichloride have been revised substantially downward and methyl chloroform has been dropped from the analysis. The report has not been altered where the Agency is currently considering, but has made no final decision regarding, changes in certain of the data used in this analysis (e.g., potency information) or in the regulatory status of certain chemicals examined.

Several additions were made to the report. A new section has been added that delineates current activities within EPA resulting from the report, including a brief discussion of the national strategy for air toxics. Air quality data have been evaluated for 1970 and the estimated risks compared to those based on 1980 data to provide a more quantitative estimate of progress under programs for criteria pollutants. Finally, data from personal exposure monitoring have been converted to aggregate national risk for several compounds to enhance comparison of the indoor/outdoor air toxics problem.

ACKNOWLEDGMENTS

Many individuals and organizations within EPA participated in this study. The report is based primarily on a series of detailed analyses and reviews done specifically for the study, often in cooperation with private companies under contract to EPA. These are listed below. We thank the authors for their efforts and for contributing so much to this analysis.

Joe Bufalini, Bruce Gay, Basil Dimitriades. "Production of Hazardous Pollutants through Atmospheric Transformations." June 1984.

Elaine Haemisegger. "Hazardous Air Pollutants: An Exposure and Risk Assessment for 35 Counties." September 1984. (Contractors: Versar; American Management Systems, Inc.)

Jim Hardin. "Issue Paper--National Air Toxics Problem: Radionuclides." August 1984. Revised verbally January 1985.

Bill Hunt, Bob Faoro, Tom Curran, Jena Muntz. "Estimated Cancer Incidence Rates for Selected Toxic Air Pollutants Using Ambient Air Pollution Data." July 1984. Revised March 1985. (Contractor: PEI)

Tom Lahre. "Characterization of Available Nationwide Air Toxics Emissions Data." June 1984. (Contractor: Radian Corp.)

Nancy Pate. "Review of the Clement Associates Report on Evidence for Cancer Associated with Air Pollution." June 1984.

Bob Schell. "Estimation of the Public Health Risks Associated with Exposure to Ambient Concentration of 87 Substances." July 1984. Revised February 1985.

Bob Schell. "Definition of the Air Toxics Problem at the State/Local Level." June 1984. (Contractor: Radian Corp.)

Vivian Thomson. "Indoor Air Pollution: Ramifications for Assessing the Magnitude and Nature of the Air Toxics Problem in the United States." July 1984.

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Donn Viviani, Doreen Sterling, Robert Kayser. "Acceptable Risk Levels and Federal Regulations: A Comparison of National Emission Standards for Hazardous Air Pollutants (NESHAP) with Other Federal Standards Based on Quantitative Risk Asessment (QRA)." May 1984.

Others within EPA provided assistance during the study.

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EXECUTIVE SUMMARY

Goals

This report summarizes the results of a study which will be used by the U.S. Environmental Protection Agency (EPA) to provide a basis for consideration of strategies to deal with the ambient air toxics problem in the United States. The study attempted to assess the magnitude and nature of the air toxics problem by developing quantitative estimates of the cancer risks posed by selected air pollutants and their sources. Four basic questions were examined:

1. What is the approximate magnitude of the air toxics problem, as measured by the estimated cancer risks associated with air pollution?
2. What is the nature of the air toxics problem, that is, what pollutants and sources appear to cause the problem and what is their relative importance?
3. Does the problem vary geographically, and if so, in what ways?
4. Are current air toxics data bases adequate? If not, what are the significant data gaps?

Context and Limitations

Readers of this report should fully understand the study's limitations so that its conclusions are interpreted correctly. The analysis was undertaken to orient EPA to the problem of airborne carcinogens, to stimulate policy discussions, and to guide further studies. Despite the fact that quantitative estimates of risk are presented in this report, the study was not initiated to support specific regulatory decisions. Instead, its goal was to obtain a quick assessment of the air toxics problem in the United States, and as such should be regarded as a "scoping" study only. Only readily available existing data were used regarding compound potencies, emissions, and ambient levels; no new data were collected. The only health effect examined quantitatively was cancer; health data on such effects as teratogenicity are not extensive enough to permit quantification. Also, acute health effects related to short-term exposures were not included.

Consideration of the limited scope of the study, as well as of the caveats and assumptions that are discussed in the text of the report, is an important responsibility of those reading and using this report. Some of the important caveats to keep in mind are identified later in the Executive Summary under "Sources of Uncertainty."

In summary, the risk estimates presented in this report should be regarded as only rough approximations of total incidence and individual risk, and should be used in a relative sense only. Estimates for individual compounds are highly uncertain and should be used with extreme caution. The reader is cautioned against applying these risk estimates to specific geographic locations, since the relative importance of particular pollutants and sources varies considerably from one place to the next.

As more data become available, these risk estimates will undoubtedly change. As such, the portrait of the air toxics problem depicted in this study should be regarded as a snapshot, the form and substance of which will certainly change as new data become available.

Analytical Methods

Three major analyses were undertaken to estimate cancer incidence and individual lifetime risks: each analysis included a separate set of compounds, with considerable overlap. The Ambient Air Quality Study used ambient data for five metals, ten organic compounds, and benzo(a)pyrene (BaP) to assess these risks. Two other analyses--the NESHAP Study and the 35-County Study--used emission estimates and exposure models to estimate incidence and maximum individual risks associated with the pollutants selected. In addition, a "BaP surrogate" approach was used to estimate cancer incidence associated with products of incomplete combustion (PIC): a dose-response coefficient relating lung cancer incidence and PIC was generated from epidemiological studies, and cancer incidence associated with PIC exposure was estimated by applying this dose-response coefficient to current ambient BaP levels. Finally, quantitative risk assessments available from other EPA activities for asbestos, radionuclides, and gasoline marketing were incorporated into the study.

Four additional reports were prepared to assist in interpreting the results of the study. One report reviewed national emissions data for over 90 compounds and provided summaries by source type, geographic location, growth trends, and data quality. Other papers were prepared on the atmospheric transformation of air pollutants, indoor/outdoor relationships for air toxics, and risk estimates used by other program offices within EPA in regulating selected toxic substances. Further, statistics on annual cancer incidence, cancer deaths, and estimates of the cancer cases associated with other causes (e.g., diet, smoking, indoor exposures) were compiled. The study team also analyzed and summarized the information available on several source categories for which current data are insufficient to perform a quantitative risk assessment. Finally, contacts made with all 50 state air pollution agencies, 33 local air pollution agencies, the Canadian government, and the Commission of European Communities revealed that virtually no other comprehensive studies are available that quantify estimated cancer incidence related to air toxics.

The study examined the magnitude and nature of the air toxics problem using existing data and, where possible, standard EPA techniques for quantitative risk assessment. We did not attempt to evaluate the validity of those techniques; rather, we tried to apply them as comprehensively as possible. For example, we relied on unit risk estimates generated by EPA's Carcinogen Assessment Group (CAG) and by Clement Associates, many of which represent plausible upper-bound estimates of unit risk. These estimates also assume 70 years of continuous exposure to outdoor ambient levels. However, where appropriate, we point out the possible effects of considering non-traditional approaches, such as examining the risks associated with indoor exposures to air pollutants.

Conclusions

Given the scope, limitations, methods, and assumptions discussed above, the following conclusions may be drawn from this study:

1. Both point sources (major industrial sources) and area sources (smaller sources that may be widespread across a given area, such as solvent usage and motor vehicles) appear to contribute significantly to the air toxics problem. Large point sources are associated with many high individual risks, while area sources appear to be responsible for the majority of aggregate cancer incidence.

2. While there is considerable uncertainty associated with the risk estimates for some substances, available data indicated that the following pollutants may be important contributors to aggregate cancer incidence from air toxics: metals, especially chromium and arsenic; asbestos; products of incomplete combustion; formaldehyde; benzene; ethylene oxide; gasoline vapors; and chlorinated organic compounds, such as chloroform; carbon tetrachloride; perchloroethylene; trichloroethylene; and vinylidene chloride.

3. A wide variety of sources contributes to individual risk and aggregate incidence from air toxics. These include: road vehicles; combustion of coal and oil; woodstoves; metallurgical industries; chemical production and manufacturing; gasoline marketing; solvent usage; and waste oil disposal. As a broad category of activities, combustion/incineration is probably the largest single source of risk.

4. For those cities with sufficient data for analysis, large city-to-city and neighborhood-to-neighborhood variation in pollutant levels and sources was found. However, our current air toxics data base is inadequate to accurately characterize most local air toxics problems.

5. Three analyses quantified estimated cancer risks due to 15 to 45 toxic air pollutants (the number of pollutants

examined varied with the different analyses). The estimates from these analyses showed a range of 5 to 7.4 cases of cancer per million people per year (1,300 to 1,700 cases annually nationwide) for the pollutants examined. These are not actual predictions of incidence, but are instead a statistical way to represent the potential health effects of human exposure to airborne carcinogens.

The reader is reminded that these estimates are highly uncertain, and is cautioned that the convergence of the various analyses on a seemingly narrow range (5 to 7.4 cases per million) is somewhat coincidental, given that estimates for individual compounds varied widely among the different analyses.

For perspective, estimated nationwide cancer cases and cancer deaths for 1983 were 850,000 and 440,000, respectively.

6. Maximum lifetime individual risks of 10^{-4} (1 in 10,000) or greater in the vicinity of major point sources were estimated for 21 pollutants, about half of those that were studied. Maximum lifetime individual risks of 10^{-3} (1 in 1,000) or greater were estimated for 13 pollutants.

7. Additive lifetime individual risks in urban areas due to simultaneous exposure to 10 to 15 pollutants ranged from 10^{-3} to 10^{-4}. These risks, which were calculated from monitoring data, did not appear to be related to specific point sources. Instead, they represent a portion of the total risks associated with the complex pollutant mixtures typical of urban ambient air.

8. Some low-production organic chemicals appeared to contribute little to aggregate incidence: 21 organic chemicals were estimated to account for a total of less than 1.0 cancer cases per year nationwide. However, this conclusion may be due in part to the lack of data concerning the emissions and toxicity of these "exotic" chemicals.

   Some of these low-production compounds did appear to be associated with high individual risks. For example, the maximum lifetime individual risk for 4,4,-methylene dianiline was estimated at 1.5×10^{-3}.

9. The study indicated that "non-traditional" sources of air toxics--such as publicly owned treatment works (POTW's) and hazardous waste treatment, storage and disposal

facilities (TSDF's)--may pose important risks in some locations. For instance, preliminary findings suggest that POTW's with industrial indirect discharges may emit volatile organic compounds in excess of 100 kkg/yr. Individual lifetime risks for a single compound at one TSDF were estimated as high as 10^{-5}.

10. EPA's criteria pollutant programs appear to have done more to reduce air toxics levels than have regulatory actions aimed at specific toxic compounds. An analysis of 16 pollutants was completed using both monitoring and emission data in order to evaluate progress made on air toxics between 1970 and 1980. The estimated cancer incidence rate for these air pollutants in 1980 was less than half that for 1970, i.e., 6.8 per million per year in 1980, compared to 17.5 per million in 1970. This seems reasonable considering the diverse array of air toxics sources, the multipollutant nature of the problem, and the relative intensity of EPA's criteria and air toxics programs.

11. Even after regulations are implemented under Section 112 of the Clean Air Act for benzene and arsenic, these pollutants still appear to make significant contributions to aggregate incidence due to air toxics. This seems to demonstrate that the base for the air toxics regulatory programs needs to be broadened to include emissions from small area sources, such as combustion, road vehicles, and solvent use.

12. Major weaknesses and gaps characterize air toxics data bases at the federal, state, and local levels. The few air toxics emission inventories available generally show inconsistencies and anomalies, the air quality data available are often inadequate to develop population exposure estimates, and few compounds have been tested adequately for health effects. The data limitations preclude performing specific comprehensive risk assessments for most urban areas, for many compounds, and for many potentially large sources of air toxics risks (such as incineration, hazardous waste disposal, indoor exposures, atmospheric transformation, and Superfund sites).

Sources of Uncertainty

Many assumptions and extrapolations are necessary to transform ambient or modeled levels of air pollutants into exposure estimates.

Whether such assumptions introduce a high or low bias into the results is difficult to assess. However, it is clear that the use of such assumptions injects a considerable degree of uncertainty into the analyses.

Some of the factors which may have led the analyses to understate the risk of cancer related to air toxics are as follows:

1. Urban ambient air is characterized by the presence of dozens, perhaps hundreds of substances. Risk estimates for most of these could not be calculated due to data limitations.

2. Indoor concentrations of certain pollutants (e.g., radon, tobacco smoke, formaldehyde, and other volatile organic compounds) are commonly several times higher than outdoor concentrations. The estimated cancer incidence associated with indoor exposures to passive smoking (500 to 5,000 annually), radon (1,000 to 20,000 annually), and with 24-hour personal exposures to 5 organic compounds (1,500 annually) indicate that indoor sources make an important contribution to air toxics risks.

3. Risks due to compounds formed in the atmosphere could not be quantified in the analyses using exposure models, but there are indications that these risks may be significant. For example, formaldehyde is formed in the atmosphere by the breakdown of other organic compounds, and some compounds (e.g., toluene) may be converted into toxic substances through photochemical reactions.

4. Although it has been shown that certain combinations of exposures may have synergistic effects (for instance, smoking and asbestos exposure), all risks were assumed to be additive.

Factors which may have caused the analysis to overestimate cancer risks associated with air toxics are as follows:

1. Cancer unit risk values were obtained from EPA's Carcinogen Assessment Group (CAG) and Clement Associates. EPA unit risk values are generally regarded as plausible, upper-bound estimates. That is, the unit risks are not likely to be higher, but could be considerably lower. In many cases, the unit risk values are preliminary.

2. The weight of evidence of carcinogenicity for the compounds examined varies greatly, from very limited to very substantial. Further, the extent of evaluation and health review performed varies considerably among compounds. For this report, a conservative scenario (i.e., that all compounds included in the report are human carcinogens) was assumed.

3. The risk assessments assume that people living in an area are exposed to the estimated ambient levels for 70 years, 24 hours a day. This especially compromises estimates of maximum lifetime individual risk. Few plants operate for 70 years, most people change their homes several times during their lives, and they also leave their neighborhoods during the day.

4. The degree to which outdoor emissions of many pollutants (e.g., trace metals) penetrate indoors is largely unknown. If emissions of a pollutant from outdoor sources do not penetrate completely indoors and if there are no indoor sources of that pollutant, then we will have over-stated risks, since we have assumed constant exposure to levels equalling those of outdoor air.

5. Although certain combinations of exposures may have antagonistic effects, all risks were assumed to be additive.

Current Activities

This study was completed and a draft was sent by the Agency through peer review late in 1984. Simultaneously, based on the preliminary findings of the analyses, EPA initiated a series of activities designed to examine the need for a new national strategy for air toxics. These included:

1. Formation of an Agency-wide Air Toxics Group to follow up on the study and to guide the development of any needed changes in national strategy.

2. Additional analytical studies to examine in more detail the controllability of the most important pollutants and the impact on risk of the current programs for criteria pollutants from now through 1995.

3. Discussions of the results of the study and possible new national strategies with all interested groups, including industry, public interest organizations, state and local governments, and legislative staffs.

Although these activities will not be completed until mid-1985, EPA is beginning to explore several changes to its program for air toxics. The focus of direct federal regulation is shifting from an emphasis on isolated large point sources to more complex situations that have greater potential for high national incidence of cancer. Generally, this will mean increased emphasis on area sources and those point sources that emit several potentially toxic pollutants. In addition, several new activities are being considered that will provide for a more comprehensive national program. These include a federal partnership with state and local agencies to evaluate and, if necessary, regulate large point sources with emphasis on reduction of high individual risk situations, and an examination of problems caused by concentrations of sources within cities or industrial regions.

I. INTRODUCTION

A number of air pollutants have been identified as having the potential to cause cancer when inhaled by humans. Section 112 of the Clean Air Act requires EPA to protect the public health from exposure to hazardous pollutants which includes such carcinogens. Recently, state officials, Congress, environmentalists, and EPA management have expressed concern about EPA's program for hazardous air pollutants. On August 26, 1983, the General Accounting Office released a report entitled "Delays in EPA's Regulation of Hazardous Air Pollutants." As a result, Congressman John Dingell, Chairman of the House Energy and Commerce Committee, called hearings held on November 7, 1983, that examined issues surrounding Section 112.

During internal discussions before the hearings, it became clear that EPA had not defined well the size or the causes of the health problems caused by exposure to air toxics. A preliminary analysis suggested that routine air releases of a group of pollutants being considered for regulation under 112 might account for no more than a few hundred cases of cancer each year. This led to some fundamental questions concerning the magnitude and nature of risks caused by air toxics.

- Do air toxics present a significant health problem?
- If air toxics do pose a significant health problem, what pollutants and sources emitting those pollutants are responsible?
- Is there an important part of the national air toxics problem that Section 112 cannot effectively address?

EPA's Deputy Administrator decided that a broad scoping study of the air toxics problem was needed before management could begin to consider changes in the national program. An ad hoc study, called the Six Month Study because of its original intended duration, was started in November 1983. Many offices and individuals within EPA contributed to this analysis, but it was primarily a cooperative effort between the Office of Air and Radiation (OAR) and the Office of Policy, Planning and Evaluation (OPPE). This report summarizes the results of that study.

In the early days of the study, we decided to emphasize four general issues that would be most useful to policymakers as they attempted to define the scope and direction of changes that may be needed to the national program for controlling toxic air pollutants.

> The magnitude of the airborne carcinogen problem.
>
> We have attempted to characterize the significance of the problem by presenting quantitative estimates of the annual incidence of cancer that may be linked to air pollution, and estimates of lifetime individual risks of cancer associated with long-term (70 years) exposure to toxic air pollutants.
>
> The nature of the problem.
>
> What air pollutants and sources emitting those pollutants cause risks to health? What is their relative significance?
>
> Geographic variability.
>
> EPA's strategy for toxic air pollutants may be influenced by geographic differences in the nature of the problem. Some sources emitting these pollutants may be relatively widespread and found in most areas of the nation. Other sources vary a great deal from city to city and controlling them may require considerable flexibility in the pollutants and sources controlled. For instance, an urban area's unique problems may not be addressed by a national regulatory program.

Adequacy of data bases.

> This study is the most comprehensive attempt to date to assemble and analyze available data on air toxics. Therefore, it is a useful vehicle for evaluating existing data bases, and identifying knowledge gaps. It should help programs set priorities and plan for future data gathering efforts, while providing policymakers with some insight into data needs for implementing a comprehensive national program.

The resources and time available required that the study be limited, in most cases, to gathering, organizing, and evaluating existing information. This suggested that the results would be less than definitive, would include major data gaps and assumptions, and would require a great deal of judgment to interpret properly. The final report supports these expectations. Risk analysis for carcinogens is very uncertain, and assessing air toxics is complicated by the poor quality of much of the available data. For several potentially significant issues, the lack of information prevents any analysis, and even in those areas with relatively good information, we had to make important assumptions. Because of these limitations, consideration of caveats, disclaimers, and assumptions is an important responsibility of those using this report. Some of the major assumptions used in this risk analysis are presented in the following section. Specific limitations associated with individual analyses are presented in the summaries of each of those studies.

This study was not conducted to support regulatory decisions on carcinogenicity or source regulation. Instead, it was designed to: (1) identify the potential and relative significance of the risks caused by pollutants and sources from a national and regional perspective; (2) set research and regulatory priorities; (3) identify those pollutants and sources that have not been well studied; and (4) develop long-term goals and general strategies for air toxics.

Because of the long list of uncertainties associated with such a study, we used several analytical approaches in attempting to assess the air toxics problem. Chapter III discusses these disparate analyses, follows with a discussion of sources and pollutants not covered by the six analyses, and then concludes with a summary of the magnitude of the problem. Chapter IV examines the nature of the airborne carcinogen problem by looking at relative contributions of pollutants and sources, geographic variability, and indirect control of air toxics. Chapter V discusses the quality and extent of available data and Chapter VI presents the study's conclusions. A final chapter summarizes Agency actions being taken as a result of the study.

II. SCOPE OF STUDY

The study has the following important characteristics:

- It is based on existing information. No new data were generated, even though existing data were used in new ways, e.g., the treatment of products of incomplete combustion. The study's value lies in assembling and organizing the available data and applying quantitative analysis to better understand an environmental problem.

- Primarily due to data limitations, the study relied solely on quantitative estimates of cancer risk associated with inhalation of ambient air. No other potential health effects were considered nor were environmental effects such as crop damage or visibility impairment.

- The only environmental pathway considered was inhalation. The study did not examine potential health risks from ingestion of air pollutants that ultimately reach humans through the diet or that are directly ingested. The study also does not address the potential environmental effects of direct deposition and urban runoff of air pollutants to surface water.

- The study focused on inhalation of ambient air. Indoor exposures were examined only for perspective.

- The study covered only 15-45 compounds and represents only a fraction of the total number of compounds present in the ambient air. The major factor preventing analysis of more pollutants is the lack of health data.

- No quantitative estimates are available for many potentially important source categories, e.g., Superfund sites, hazardous waste disposal, and pollutants formed in the atmosphere.

- The study focused primarily on expected, routine, and continuous emissions of hazardous substances. Accidental releases, such as those that were responsible for the tragedy in Bhopal, India, as well as smaller, more common releases, were not the subject of our study. However, the ambient air quality analysis may have included the accidental releases that contribute to ambient concentrations of some compounds.

- The risk estimates in the report are based on layers of assumptions concerning the health effects of chemicals, the degree of human exposure, and the way these substances interact inside the human body. Many of them are conservative, while others may tend to underestimate risk. The result is a degree of uncertainty that we cannot even begin to quantify. The numerical estimates presented in this report should be viewed as a rough indication of the potential cancer risks caused by a few pollutants. Many of the absolute values are almost certainly inaccurate, and the strongest and best use of the numbers is in making relative comparisons across pollutants and sources, and in setting priorities and allocating resources.

A. Reasons for Assessing Only Cancer Risk

At the beginning of the study, we decided to rely solely on estimates of cancer risk associated with air pollutants and exposures. There were several reasons:

- Cancer is a significant cause of death in the United States: approximately 20 percent (440,000 per year) of all deaths in the United States are caused by cancer.

- Urban areas have higher lung cancer rates than rural areas.
- Several identified air pollutants are human carcinogens, e.g., benzene, arsenic, and vinyl chloride.
- The public is concerned about the possible link between environmental pollution and cancer incidence.
- The use of a nonthreshold assumption in estimating cancer risk has broad scientific support. Therefore, even the very low concentrations typical of ambient air may still be a problem. Most acute and subchronic health effects appear to have a threshold.
- Cancer incidence lends itself to probabilistic analysis. There is a well-established mathematical model, i.e., the Crump model, for estimating risk at low doses. This is not the case for other effects.
- There are short-term indicators of mutagenicity/carcinogenicity, e.g., the Ames test.

The ambient concentrations of most noncriteria pollutants appear generally to be below the threshold levels associated with most acute and subchronic health effects. Most such effects are caused by exposures to concentrations in the parts per million range, while ambient concentrations of most compounds are in the parts per billion range. However, this may not always be the case. The ambient concentrations near some sources may approach the levels associated with acute health effects. The lack of data on acute health effects is a serious omission in this report, and these health effects should not be ignored when assessing an air toxics source.

Compared to cancer and acute effects, there is even less information available concerning mutagenicity and teratogenicity. There are a few examples of compounds with data. Ethylene dibromide and ethylene oxide have been shown to be mutagenic in test systems, and 2,3,7,8-TCDD has been shown to be a developmental toxin in animals. Unfortunately, the data for most compounds are too limited to qualitatively determine whether the substances are potentially mutagenic or teratogenic. For those few substances with sufficient weight of evidence, there is rarely enough information to develop any reliable dose-response estimates. While it is generally accepted that there are thresholds for some teratogenic effects in test animals, the data are seldom sufficient to calculate those threshold levels.

The uncertainty is compounded when animal data are used to predict human teratogenic effects. For teratogens, there tend to be multiple end points, and the timing of exposure is often crucial. These parameters may not be the same for both animals and humans.

In contrast, there is biological support for a nonthreshold theory of carcinogenicity in both animals and humans. Furthermore, it is generally accepted that a substance that causes cancer in test animals is likely to be carcinogenic to humans as well. This has not been established for other health effects. Given these gaps in our knowledge, quantitative risk assessments are most defensible for cancer. More work is needed to establish models and methods for assessing quantitatively the risk of other health effects.

This study did not analyze the impact of major accidental releases of toxic substances such as MIC in Bhopal, India. These clearly are a major issue, but were outside the scope of this report. However, EPA recognizes the potential of such releases and is currently examining regulatory options.

III. METHODS USED TO ESTIMATE CANCER RISK

Assessing the cancer risks of exposure to an environmental pollutant requires three pieces of information: (1) an estimate of the carcinogenic potency (the unit risk value) of the pollutant being considered; (2) an estimate of the ambient concentration that an individual or group of people may breathe; and (3) an estimate of the number of people that are exposed to those concentrations.

This study is based on methods for assessing carcinogenic potency and estimating ambient concentrations now in use throughout EPA. We did not judge the appropriateness of these methods, nor did we attempt to use other methods. We thought that a comprehensive analysis of risk assessment techniques was beyond the scope of this study, and using other methods would make risk comparisons with other EPA programs more difficult. The following is a discussion of the methods that we used and the assumptions upon which our estimates are based.

A. Estimating the Carcinogenic Potency of Pollutants

Assessing the risk of cancer caused by exposure to toxic substances in the environment is a complex, controversial, and uncertain business. For most of the pollutants covered by this analysis, the estimates of risk per unit dose were developed by EPA's Carcinogen Assessment Group (CAG).[1] To calculate such estimates, CAG made the following major assumptions:

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[1] Quantitative estimates of carcinogenic potency (the unit risk value) are expressed as the chance of contracting cancer from a 70-year lifetime exposure to a concentration of 1 ug/m^3 of a given substance. Generally, the unit risk value represents the probability of cancer cases, not deaths. However, since the epidemiological studies that generated the potency number for PIC (products of incomplete combustion) are based on lung cancer mortality, the PIC estimates used in this report imply lung cancer deaths.

- CAG uses experimental data showing that a substance is carcinogenic in animals to demonstrate that the substance may be carcinogenic in humans as well.

- In the absence of human data, CAG uses the results of such animal bioassays to estimate the probability of carcinogenic effects in humans. Such extrapolations assume humans to be as sensitive as the most sensitive animal species tested.

- CAG uses a nonthreshold, multistage model that is linear at low doses to extrapolate from high-dose response data (either occupational studies or animal bioassays) to the low doses typically caused by exposure to ambient air. In other words, CAG assumes that carcinogenic substances cause some risk at any exposure level. These unit risk values represent plausible upper bounds--i.e., they are unlikely to be higher, and could be substantially lower.

- CAG assumes that exposed individuals are represented by a reference male having a standard weight, breathing rate, etc. No reference is made to health, race, nutritional state, etc.

Some people have charged that some of these assumptions overstate risk. However, other factors may offset the conservatism in the techniques.

- People are exposed to complex mixtures of chemicals. Data are not available to demonstrate or deny the existence of either synergistic or antagonistic health effects at low exposures.

- Virtually all animal and human data are based on exposure to adults. There may be enhanced risk associated with fetal, child, and/or young adult exposures to some agents.

- There may be high susceptibility for some population groups because of metabolic differences or inherent differences in their response to the effects of carcinogens.

The Administration recently took a position on some of the more controversial assumptions above. On May 22, 1984, the White House Office of Science and Technology Policy (OSTP) released its final report, Review on the Mechanisms of Effect and Detection of Chemical

Carcinogens. The report's statement of principles concludes that available information "does not allow one to define the existence or location of a threshold" for carcinogenicity. Furthermore, the principles state that "a model which incorporates low-dose linearity is preferred when data and information are limited as is the usual case and when much uncertainty exists regarding the mechanisms of carcinogenic action."

B. Estimating Exposure to Pollutants

For most of the analyses summarized in this report, two measures of risk were calculated: liftime individual risk and estimated annual incidence. Lifetime individual risk is a measure of the probability of an individual's developing cancer as a result of exposure to an ambient concentration of an air pollutant or group of air pollutants over a 70-year period. Often, the maximum lifetime individual risk is also presented, which usually applies to people living nearest the source.[2] In an attempt to gauge the significance of additive risks, we also calculated multipollutant individual risks caused by many pollutants measured in the same area. These multipollutant risks were not associated with a specific point source.

Aggregate or population risk estimates, on the other hand, are estimates of the annual incidence of excess cancers for the entire affected population. These estimates are calculated by multiplying the estimated concentrations of the pollutant by the unit risk value and by the number of people exposed to different concentrations. This calculation yields an estimate of the total number of excess cancers that may occur over a 70-year period. The total must then be divided by 70 to estimate annual incidence.

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[2] A maximum individual lifetime risk estimate of 3.0×10^{-4}, for example, near a point source implies that if 10,000 people breathe a given concentration for 70 years then it is likely that three of the 10,000 will develop cancer as the result of the exposure to that pollutant from the source.

C. Using Monitoring Data to Estimate Ambient Concentrations

This study used two major techniques to estimate the ambient concentrations of pollutants that people may inhale. One technique used measurements of ambient air quality and the other relied on emission estimates and dispersion modeling. Each technique has advantages and drawbacks. Using direct measurements of ambient concentrations to estimate risk avoids the problems of incomplete emission inventories, incomplete knowledge on current control status, a lack of knowledge concerning pollutants formed or destroyed in the atmosphere, and the list of errors associated with dispersion modeling. However, there is significant potential for error in using monitoring data to estimate risk.

The most important potential source of error is the classic problem of extrapolating measurements at a single site to a much larger geographic area in order to estimate population exposure. To estimate concentrations in a city, we were forced to average measured values and assume that these values applied to the entire area. The number of monitoring sites in a metropolitan area ranges from one or two to a maximum of ten in Baltimore and

Philadelphia. Because of this limited coverage and because monitors are often intentionally located away from major sources, using monitoring data probably is especially unsuitable for estimating maximum individual risk.

In addition, estimating annual incidence forced us to extrapolate the available data for a relatively small number of areas to the rest of the nation. For trace metals and organic particulates, the National Air Monitoring System and State and Local Air Monitoring Systems (NAMS/SLAMS) contain data for counties representing a total of 25 million to 75 million people. Data on volatile organics are available for areas with a total population of only 2 million to 25 million people. The ambient air quality study made certain assumptions in extending these data to the rest of the nation. We have no way to assess the accuracy of these methods which used ambient data measured at certain spots to estimate concentrations elsewhere.

Third, because cancer risk assessment assumes long-term exposures, the most useful data are long-term average concentrations, preferably annual averages. Very few studies have collected ambient samples for toxics continuously for an entire year. For this study, monitoring data for 20 days a year was labeled as being sufficient for calculating an annual average. This was available for most of the trace metals. For organics, annual averages were calculated if monitoring data were available for ten separate days spread over at least two quarters.

Finally, all air quality data are subject to errors in sampling and analytical methods. These are serious problems for many noncriteria pollutants. For many compounds, good analytical methods have yet to be developed.

D. Using Emission Estimates and Dispersion Modeling to Estimate Ambient Concentrations

Several of the analyses presented in this report relied on emission estimates and dispersion modeling to estimate ambient concentrations. A major advantage of this method over ambient data is the ability to characterize the contribution of various sources. Also, emission modeling provides more comprehensive geographical coverage and, therefore, can identify "hot spots" that are of concern because of high individual risk. Finally, modeling generally allows a larger number of pollutants to be considered, and it avoids the problem of geographic extrapolation.

Emission estimates and dispersion modeling were used in most of the analyses summarized in this report. The major ones are the 35-County Study, the NESHAP Study, and work completed by the Office of Radiation Programs on radionuclides. Conceptually, the models all operate the same way. Emission estimates for area and mobile sources are apportioned uniformly over the entire area being considered, while point sources are located at a specific site. Emission estimates for point sources are developed using available sources of information, which may vary widely in quality. The emission estimates are loaded into the computer dispersion model, along with information on stack height and diameter, emission velocity, and temperature. Meteorological data (wind speed, direction, and stability) from the nearest of over 300 National Climate Center sites are entered into the model, along with population distribution information from 1980 census data. Running the models results in estimates of ambient concentrations at different distances from the source. The dispersion models were run for 50 km in the 35-County Study, 20-50 km in the NESHAP analysis depending on the pollutant, and 80 km for the radionuclides analysis. The appropriate choice for the outer boundary when estimating pollutant dispersion is a matter of considerable debate.

Some of the major issues surrounding the use of both monitoring and dispersion modeling techniques used in this study to estimate exposure are as follows:

- The dispersion models assume flat terrain and average meteorological conditions. Rough terrain in the area surrounding a source, such as a valley, would probably cause higher concentrations near point sources and lower concentrations further away from the source.

- Although exposure estimates apply to a certain point in time, our risk assessments assume that the people who live in an area are exposed to the estimated ambient concentrations for 70 years. In other words, we assume that the plant operates for 70 years, that no one moves in or out of an area, and that no one moves around within the area. Few plants operate for 70 years, and most people change homes several times during their life. However, a person may still be exposed to emissions of the same or different toxic compounds after moving from an area.

- A related issue is the assumption that people are continually exposed to outdoor ambient concentrations of pollutants. In fact, most Americans spend 80 percent to 90 percent of their time indoors. Thus, a significant part of total exposure to air toxics occurs indoors. We were unable to quantify the risks from indoor exposures to most of the substances examined in this study. However, there are strong indications that indoor levels of many volatile organic compounds are higher than outdoor levels, since there are many indoor sources of organic compounds.

No indoor/outdoor comparisons were found for the metals examined in this study, but the limited data available for other trace metals show that indoor air levels are sometimes higher and sometimes lower than outdoor levels.

- Dispersion modeling is often extended to only 20 km from the source. This technique can lead to understating risk if extending dispersion increases significantly the number of people exposed. To see what difference a 50 km boundary would make, five organic substances were modeled to that distance. This change increased annual cancer incidence by a factor of 1.35.
- Dispersion estimates are rarely based on site-specific meteorology. Often, data from hundreds of kilometers away must be used.
- In running the dispersion models, we do not consider increases in concentrations that could result from reentrainment of toxic particles from streets, rooftops, etc. With the exception of radionuclides, we also do not consider background concentrations and emissions from other sources not explicitly included in the analyses, including toxics formed in the atmosphere.
- Emission estimates are generated using data and assumptions that could be in error. For example, although the 35-County Study incorporates plant-specific emission estimates whenever possible, the pollutant releases for the remaining sources were developed by applying speciating factors against the VOC and TSP data in the National Emission Data System (NEDS). Unfortunately, some of the information in NEDS is of questionable consistency and quality for the purposes of quantitative risk assessment.
- For other analyses, estimates are based on plant capacity and emission factors. These studies assume that plants continuously operate at an assumed percentage of capacity and that no changes in emission rates occur. Emissions from malfunctions and upsets were not considered in this study.

IV. MAGNITUDE OF THE AMBIENT AIR TOXICS PROBLEM

A. Introduction

One of the major goals of this study was to improve our understanding of the size of the overall public health problem caused by air toxics, a task that has been colorfully characterized in the trade press as determining whether the air toxics problem is "an elephant or a mouse." We identified several analytical techniques for assessing the nature and magnitude of the air toxics problem. Each method offered different advantages, as well as varying degrees of resolution and uncertainty. Rather than select one approach for analyzing such a complex issue, we chose to complete several studies:

- An assessment of the hazardous air pollutant problem based on state and local experience;
- An estimate of national exposure and risk from about 40 pollutants being considered for listing under Section 112 of the Clean Air Act;
- A more detailed estimate and analysis of exposure and risk in 35 counties for about 20 pollutants, including consideration of sources that were not considered sources of air emissions in the past, such as municipal sewage treatment plants (POTWs) and waste oil combustion;
- An analysis of existing ambient air quality data; and
- Risk estimates for pollutants and sources either not covered by the analyses above--e.g., radionuclides, asbestos, and gasoline marketing--and a discussion of others not easily quantified--e.g., dioxin and combustion of hazardous waste in boilers.

In this chapter, we describe each of these studies in more detail and express the magnitude of the problem in three ways: annual national cancer incidence; annual incidence per million people; and lifetime individual risk. We then summarize and compare the results from each effort, and develop general conclusions.

Again, we must caution against misuse of the results of this scoping study. The analysis was not undertaken to lead directly to decisions on carcinogenicity nor regulation. It was designed to: (1) identify the potential significance of the risk caused by air toxics from a national and regional perspective; (2) assist the Agency in setting research and regulatory priorities; (3) identify those pollutants and sources for which only scant data exist and should therefore be explored in more detail; and (4) assist in developing long-term goals and general strategies for air toxics.

B. Summaries of Individual Analyses

1. Survey of State and Local Agencies, Canada, and Europe

The responsibility of dealing with air toxics is not unique to EPA or to the United States. Many state and local agencies have active air toxics programs; also, other industrialized nations have the same public concern over environmentally related cancer as the United States. We reasoned that they may have the same need as EPA to define the risks from air toxics in order to justify programs and to set priorities. Therefore, a portion of the study involved communication with Canada, the European community, all states, and 33 major local air agencies regarding their risk assessment activities. [3,4,5]

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[3] Memorandum from B.J. Steigerwald (U.S. EPA) to Alan Jones *et al.*, (U.S. EPA), "Air Toxics Program in Canada," April 16, 1984.

[4] Memorandum from Delores Gregory (OIA) to B.J. Steigerwald (U.S. EPA), "E.C. Regulation of Hazardous Air Pollutants," May 3, 1984.

[5] Radian Corporation, "Definition of the Air Toxics Problem at the State/Local Level," EPA Contract 68-02-3513, Work Assignment 45, June 1984.

Of the agencies and organizations contacted, only California has attempted to quantify public health risks from air toxics. Officials in Canada believe that risk assessment will be increasingly important in their toxics programs, but they have not yet developed methods and do not apply risk assessment systematically. They will evaluate in detail the results of this study. We sent cables to the Commission of the European Communities through EPA's Office of International Activities and discussions were held with individuals involved in toxics programs in Europe. There is much information available from the international community on the potential toxicity of various compounds, but nothing seems to be available on cancer incidence or individual risks from exposure to ambient air pollution.

The California estimate was an isolated analysis published in 1982 to support proposed regulation on air toxics.[6] It used Los Angeles air quality data for nine specific compounds to calculate excess lifetime cancer rates per million population. Potency for each compound was determined in a unique way, using an air equivalent of EPA's Water Quality Criteria, rather than the unit risk value used in EPA's risk assessment procedures. Therefore, the results are not directly comparable to the results we obtained in this study. For the 9 compounds selected, the California analysis estimated about 1,000 lifetime cancers per million people, or about 14 annual cases per million. The study was used by the California

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[6] Batchelder, J. et al., "Proposed Amendments to Chapter 1, Part III of Title 17, California Administrative Code, Regarding the Emission of Toxic Air Contaminants," California Air Resources Board, September 1982.

Air Resources Board for orientation purposes only and to show that the problem deserved additional attention. The Board does not recommend the study be given weight beyond its original purpose.

Since most state and local agencies included in the poll expressed concern over air toxics but could not quantify their concern directly, we explored other more subtle indicators of the problem. Counting air "episodes," "incidents," or "complaints" involving health scares produced no usable statistics. An evaluation of source permits indicated that, at least for states with fenceline ambient standards, air toxics programs could utilize substantial Agency resources and often require controls beyond those needed for criteria pollutants.

For example, Michigan issues about 1,000 new source permits a year for emissions of toxic pollutants. New York reviews 36,000 operating permits every 1 to 5 years under their air toxics regulation; this number increased by 6,000 emission points in the past 2 years. Each year, Illinois reviews 5,000 to 6,000 permits that involve emissions of air toxics. In a recent detailed study of 42 permits for source categories likely to emit toxics, Illinois found that 20 of the sources were required to apply controls beyond those needed for criteria pollutants.

In summary, our analyses could not find any other study that has attempted to comprehensively define risks from air toxics. However, general concern about the problem is universal, and an increasing number of states have begun to issue air toxics permits to large numbers of new and existing sources. Although sometimes based on use of best technology, these permits are generally based on dispersion modeling and compliance with fenceline ambient standards that are derived from occupational guidelines.

2. Evaluation of Epidemiological Studies Linking Cancer with Air Pollution

Background

The traditional way to demonstrate the effect of environmental pollution on public health has been to perform an epidemiological study. A variety of such studies has been attempted for air pollution. Our primary source of data on these studies was a Clement Associates report for EPA that described and critically evaluated the evidence for cancer associated with air pollution.[7,8]

The report assembled three main types of evidence linking cancer incidence to air pollution: epidemiological studies, laboratory studies on the mutagenicity of airborne materials, and ambient air monitoring data for pollutants known to be carcinogens. Data from the mutagenicity and monitoring studies confirmed other reports that extracts of airborne material from polluted air and emissions from motor vehicles and stationary sources are mutagenic or carcinogenic in experimental bioassay systems.

The report also reviewed epidemiological studies linking air pollution and lung cancer by using levels of benzo(a)pyrene (BaP), a known potent carcinogen, as an indicator of air pollution.[9]

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[7] Clement Associates, Inc., "Review and Evaluation of Evidence for Cancer Associated with Air Pollution," (EPA-450/5-83-006) Review Draft, November 9, 1983.

[8] Pate, Nancy, "Review of the Document 'Review and Evaluation of the Evidence for Cancer Associated with Air Pollution' and Assessment of This Approach for Better Defining the Extent and Magnitude of the Air Toxics Issue," June 1984.

[9] BaP is a ubiquitous pollutant generally found in emissions from incomplete combustion processes, especially of wood and coal in small combustion units and in motor vehicle exhaust. BaP is one of the literally hundreds of organic particulates known as polynuclear organic compounds. Many polynuclear organics are carcinogenic, many are not.

Using selected of these studies, the Clement report presented calculations of the number of lung cancer deaths which could be associated with a given level of air pollution as characterized by BaP concentrations. By combining lung cancer mortality statistics from the 1960s with estimated levels of BaP in the 1930s and 1940s, Clement estimated that roughly 10,000 cases of lung cancer per year during the 1960s were attributable to air pollution. Unfortunately, because of the long lag time between exposure and onset of cancer, these findings are not directly relevant to the hazard posed by current air pollution, particularly since BaP concentrations have generally declined by a factor of 10 since the 1960s.[10]

Despite this limitation in the direct use of the results of epidemiological studies, we decided we could not ignore the polynuclear organics represented by BaP in this analysis. Even though overall BaP emissions have decreased significantly since the 1930s and 1940s, BaP-related compounds are still present in the ambient air and may still represent an important part of the air toxics problem. For example, a recent study completed in New Jersey examined ambient BaP concentrations and mutagenicity of organics extracted from inhalable particulate matter samples. BaP levels and the mutagenicity of the particulate increased significantly during the winter relative to the summer.[11]

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[10] Pate, Nancy, "Review of the Document."

[11] Lioy, Paul J., and Daisey, Joan M., "The New Jersey Project on Airborne Toxic Elements and Organic Substances (ATEOS): A Summary of 1981 Summer and 1982 Winter Studies," Journal of the Air Pollution Control Association, Volume 33, Number 7, July 1983.

Thus, we decided to use a dose-response coefficient derived from data cited in the Clement report, and to combine it with current air quality data and estimates of BaP emissions to estimate cancer incidence associated with the large category of BaP-related pollutants which we will refer to in this study as Products of Incomplete Combustion (PIC). The Clement report presented 14 estimates obtained from 12 separate reports of the dose-response relationship between air pollution levels as indexed by BaP concentrations and lung cancer rates. Of these 14 estimates, 6 were derived from occupational epidemiological studies, while 8 were derived from general population studies that related cancer deaths in the period 1959-1975 to ambient BaP levels from 1958-1969.

Clement Associates adjusted the dose-response coefficients in these general population studies downward to account for the decline in ambient BaP levels during the lag periods between exposure and death from lung cancer. In accordance with recommendations by research groups within EPA, certain of the occupational dose-response estimates presented in the Clement report were revised (for example, the Carcinogen Assessment Group's latest estimate for coke oven emissions was substituted for that appearing in the Clement report). The final potency estimates (as expressed by lung cancer deaths per year per ng/m^3 BaP) for the occupational studies varied from 0.09 to 0.80 x 10^{-5}, whereas those for the general population studies varied from 0.3 to 1.4 x 10^{-5}. Averaging the potencies for each of the two categories of studies yielded estimates of 0.7 x 10^{-5} (general population) and 0.5 x 10^{-5} (occupational). The midpoint of these two values--0.6 X 10^{-5} deaths per ng/m^3 BaP per year (or 0.42 deaths per 70 years per ug/m^3 BaP)--was selected and combined with estimates of population exposure to BaP. Based on air quality data, 610 incidences of lung cancer per year nationwide were estimated to be attributable to PIC, whereas 124 deaths per year were estimated using BaP emission data and the more limited population studied in the 35-County Study.

There are several key limitations to using BaP levels as a surrogate for exposure to a complex mixture of compounds, as we have done in this analysis. A major weakness of using the potency estimates derived from the occupational studies is that the mix of PIC in the exposures studied (coke oven emissions, roofing tar fumes, and gas fumes) almost certainly differs from that of the ambient air. Limitations of general population studies are that BaP in these studies is used as a surrogate for all air pollution involved in lung cancer, not just PIC, and also that BaP ambient levels in the 1930s and 1940s had to be estimated. In addition, the proportion of carcinogenic activity attributable to BaP in PIC mixtures is known to vary among source categories and sometimes within a source category (e.g., among different automobiles). The impact of this varying ratio of BaP to other compounds is further complicated since synergistic and antagonistic effects between BaP and other PIC compounds are known to occur, but at present are unquantifiable. All of these factors indicate strongly that BaP is almost certainly not a stable index of the carcinogenicity of polluted air.

In spite of the limitations of the BaP-surrogate method, we could find no better alternative for estimating risk due to PIC.

Simply citing risk estimates for mixtures from specific sources of PIC was not an option, since quantitative risk estimates are available for only one source category of interest--coke oven emissions--which comprises only a small fraction of total estimated PIC emissions. Also, sufficient data on potency and emissions do not exist to characterize PIC risks on a compound by compound basis. There are precedents for using BaP as a surrogate in just this way. The National Academy of Sciences (NAS) recently used BaP as a proxy to estimate the cancer risk from polycyclic aromatic hydrocarbons (a chemically defined analogue of our more loosely defined "PIC"). In a 1983 report entitled "Polycyclic Aromatic Hydrocarbons: Evaluation of Sources and Effects," the NAS estimated cancer risks as follows:

> This appendix...assumes that benzo(a)pyrene (BaP) can be used as a proxy for PAH's and that human exposure to BaP in the ambient air at an average concentration of 1 ng/m^3 over an entire lifetime has the effect of increasing by 0.02-0.06% the risk of dying prematurely (at or before the age of 70) because of lung cancer. Although the appropriateness of BaP as a surrogate for PAH's in general has been questioned, it has been so used extensively in the past, and much of the available information refers to it as an indicator for exposure to PAH's. (p. D-1)

By way of comparison with the potency estimate used in our analysis (0.6×10^{-5}), the NAS report's estimates of lifetime potency translate into 0.3 to 0.9×10^{-5} lung cancer deaths per year per ng/m^3 BaP. The fact that the midpoint of this range was identical to the potency chosen for our analysis gave us greater confidence in the use of the potency.

The same NAS report presented estimates of cumulative lung-cancer incidence due to lifelong exposure to 1 ng/m^3 BaP from both gasoline- and diesel-fueled vehicles (accompanied by other compounds in the ratios produced by the source). These estimates varied from a low of 20 per 100,000 for the single gasoline-fueled vehicle examined to a high of 787 per 100,000 for a diesel-fueled vehicle, compared to that of 43 per 100,000 for coke oven emissions. The PIC-surrogate approach used in the 35-County Study assumes all sources have the same incidence per ng/m^3 of BaP. In contrast, the lung-cancer incidence for coke oven emissions is 10-200 times greater than that for the gasoline- and diesel-fueled vehicles, when expressed on a constant-weight-of-extract basis, rather than a constant-weight-of-BaP basis. This indicates that BaP is not a good surrogate for PIC associated with particulate emissions from road vehicles.

Thus, we acknowledge that there are real analytical problems associated with estimating risk due to PIC and that there is variation in the BaP-surrogate potency estimates. However, since this report was intended to focus policy and planning activities and was not meant to serve as the basis for regulatory acton, we decided to include the incidence estimate for PIC as a preliminary estimate of the magnitude of the PIC problem.

3. NESHAP Study

Background

The NESHAP Study was one of two major analyses that employed dispersion modeling to assess exposure and risk due to air toxics.[11] EPA's Human Exposure Model was employed to convert point source emission estimates (routine emissions, not accidental) into estimated ambient levels. The study was designed to examine in more detail the growing belief that sources covered in the past under EPA's NESHAP regulatory program (i.e., industrial producers and major users of the chemicals of concern) may be responsible for only a small part of the air toxics problem. The risk estimates obtained in this study are national in scope, and consider emissions obtained from traditional air pollution inventories. The sources covered included mobile and area sources, but the emphasis was on large point sources. This analysis did not consider some potentially important pollutants, such as radionuclides, gasoline vapors and products of incomplete combustion (PIC), and such non-traditional sources as POTWs and hazardous waste disposal.

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[11] Schell, R.M. "Estimation of the Public Health Risks Associated with Exposure to Ambient Concentrations of 87 Substances," OAQPS, U.S. EPA, July 1984. Revised February 1985.

The original intent of this effort was to estimate exposure and risk for 87 pollutants: the original 37 candidates for listing under Section 112 and 50 additional substances identified by EPA's Office of Air Quality Planning and Standards (OAQPS). OAQPS identified this latter grouping of pollutants using the Hazardous Air Pollutant Prioritization System (HAPPS) developed by Argonne National Laboratories. OAQPS also considered ambient air monitoring data and production data in developing the list. Unfortunately, after a great deal of effort to gather all available dose-response data on these pollutants, we were only able to quantitatively analyze 42 compounds (see Table 1). The qualitative judgment regarding the carcinogenicity of some of these compounds is still an open question: such compounds are included here for analytical purposes only. All of the unit risk values used in this report are presented in Attachment A.

Emission estimates for 27 of the 42 compounds were developed using OAQPS staff analyses and other OAQPS contract documents. For the remaining 15 compounds little information was available. Surrogate estimates of exposure were made for these using a "best-fit" approach with known compounds based on physical properties, uses, and production volumes.

Findings

For the 42 compounds included in the NESHAP analysis, a total nationwide annual cancer incidence of 504 was calculated (see Table 1). Roughly 90 percent of these can be attributed to the following 8 compounds, ranked in descending order: chromium; ethylene oxide; benzene; trichloroethylene; ethylene dibromide;

TABLE 1

NESHAP STUDY: PRELIMINARY APPROXIMATION OF ANNUAL INCIDENCE AND MAXIMUM LIFETIME RISK

Pollutants Having Some Evidence of Carcinogenicity*	Preliminary Approximation of Maximum Individual Lifetime Risk**	Preliminary Approximation of Incidence**
Acrylamide	$7.4x10^{-5}$	0.01
Acrylonitrile	$3.8x10^{-3}$	0.42
Allylchloride	$1.3x10^{-6}$	<0.01
Arsenic	$6.5x10^{-3}$	4.70
Benzene	$8.0x10^{-3}$	32.30
Benzylchloride	$3.0x10^{-5}$	<0.01
Beryllium	$1.0x10^{-4}$	1.20
1,3 Butadiene	$9.7x10^{-6}$	0.01
Cadmium	$3.6x10^{-3}$	8.50
Carbon tetrachloride	$5.8x10^{-4}$	14.00
Chloroform	$3.0x10^{-3}$	0.27
Chromium†	$1.6x10^{-1}$	330.0
Coke oven emissions	$2.0x10^{-2}$	8.60
Diethanolamine	$2.0x10^{-7}$	<0.01
Dimethylnitrosamine	$5.4x10^{-5}$	0.05
Dioctyl phthalate	$9.8x10^{-6}$	<0.01
Epichlorohydrin	$1.9x10^{-6}$	<0.01
Ethyl acrylate	$4.7x10^{-5}$	<0.01
Ethylene	$4.9x10^{-4}$	<0.01
Ethylene dibromide	$1.6x10^{-4}$	26.70

* The weight of evidence of carcinogenicity for the compounds listed varies greatly, from very limited to very substantial. Further, the extent of evaluation and health review performed varies considerably among compounds. However, for the purposes of this report, a conservative scenario (i.e., that all compounds examined could be human carcinogens) has been assumed.

** Because of the uncertainties in the data used to make these estimates, they should be regarded as rough approximations of total incidence and maximum lifetime individual risk. Estimates of incidence for individual compounds are much less certain. These incidence and maximum risk estimates have been performed to provide a rough idea of the possible total magnitude of the air toxics problem, and will be used only for priority-setting and to provide policy guidance.

† Risk estimates assume that all species of chromium are carcinogenic, although only certain species have evidence of carcinogenicity. Current data do not allow differentiation among species.

TABLE 1 (cont.)

NESHAP STUDY: PRELIMINARY APPROXIMATION OF ANNUAL INCIDENCE AND MAXIMUM LIFETIME RISK

Pollutants Having Some Evidence of Carcinogenicity*	Preliminary Approximation of Maximum Individual Lifetime Risk**	Preliminary Approximation of Incidence**
Ethylene dichloride	$3.6x10^{-3}$	0.92
Ethylene oxide	$6.8x10^{-3}$	47.80
Formaldehyde	$6.1x10^{-4}$	1.60
4,4 Isopropylidenediphenol	$1.1x10^{-6}$	0.03
Melamine	$1.5x10^{-6}$	<0.01
Methyl Chloride	$1.2x10^{-5}$	<0.01
Methylene chloride	$9.0x10^{-6}$	1.0
4,4-methylene dianiline	$1.5x10^{-3}$	0.02
Nickel subsulfide	$8.3x10^{-5}$	0.02
Nitrobenzene	$1.2x10^{-6}$	<0.01
Nitrosomorpholine	$6.0x10^{-9}$	<0.01
Pentachlorophenol	$1.7x10^{-5}$	0.12
Perchloroethylene	$4.6x10^{-4}$	2.90
PCBs	$3.0x10^{-4}$	0.21
Propylene dichloride	$2.1x10^{-6}$	<0.01
Propylene oxide	$3.0x10^{-2}$	0.97
Styrene	$3.3x10^{-5}$	<0.01
Terephthalic acid	$1.5x10^{-6}$	<0.01
Titanium dioxide	$3.2x10^{-7}$	0.01
Trichloroethylene	$1.0x10^{-4}$	9.70
Vinyl chloride***	$3.8x10^{-3}$	11.70
Vinylidene chloride	$4.2x10^{-3}$	0.04
Total		503.8

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* The weight of evidence of carcinogenicity for the compounds listed varies greatly, from very limited to very substantial. Further, the extent of evaluation and health review performed varies considerably among compounds. However, for the purposes of this report, a conservative scenario (i.e., that all compounds examined could be human carcinogens) has been assumed.

** Because of the uncertainties in the data used to make these estimates, they should be regarded as rough approximations of total incidence and maximum lifetime individual risk. Estimates of incidence for individual compounds are much less certain. These incidence and maximum risk estimates have been performed to provide a rough idea of the possible total magnitude of the air toxics problem, and will be used only for priority-setting and to provide policy guidance.

*** NESHAP being applied to many vinyl chloride sources.

carbon tetrachloride; coke oven emissions; and cadmium.[13] Maximum individual risks of 10^{-3} or greater were estimated for 13 compounds: acrylonitrile; arsenic; benzene; cadmium; chloroform; chromium; coke oven emissions; ethylene dichloride; ethylene oxide; 4-4 methylene dianiline; propylene oxide; vinyl chloride; and vinylidene chloride.

In addition to the usual uncertainties associated with risk assessment, there are further complications with the risk estimates for several compounds, including chromium, carbon tetrachloride, and formaldehyde. These considerations demonstrate the need for caution in interpreting such studies.

In the case of chromium, only the hexavalent form has been proven to be carcinogenic, although it is a potent carcinogen. There is now insufficient evidence to determine that the trivalent form is also carcinogenic. The NESHAP analysis, however, assumes that total chromium releases are carcinogenic and that trivalent chromium is as potent as hexavalent. There is no information now available on the ratio of trivalent to hexavalent for emissions or ambient concentrations, but some occupational exposure studies suggest that the trivalent form may dominate in some source categories. On the other hand, several important source categories are known to emit at least some hexavalent chromium, and there is some evidence that changes in the valence state can occur in the atmosphere. The problem of speciation adds one more layer of uncertainty to the risk estimates for chromium.

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[13] Approximate individual percentage contributions of some of the more important compounds are: chromium (65%); nickel (70%); ethylene oxide (10%); benzene (70%); ethylene dibromide (5%); coke oven emissions (2%); cadmium (2%); and carbon tetrachloride (3%).

Carbon tetrachloride is a very stable organic compound that has a half-life of about 35 years, compared with a half-life of hours or days for most other common volatile organic compounds. As a result, carbon tetrachloride is accumulating in the atmosphere. Therefore, current emissions are associated with current and future cancer risks. The NESHAP analysis covers only current risks and estimates incidence at 14 per year. If current ambient levels (rather than modeled levels) are used, the incidence estimate increases to about 85 per year. Carbon tetrachloride also has the potential to deplete stratospheric ozone and thereby to indirectly increase the incidence of skin cancer. For example, preliminary calculations estimate that by the year 2020 U.S. emissions of carbon tetrachloride could be responsible for between 500 and 22,000 cases of skin cancer annually in the U.S., resulting in 3 to 220 deaths per year.[14]

Finally, formaldehyde is another example of the complexities that exist in the analysis. It can be formed in large quantities in the atmosphere, and the risks posed by the resulting ambient concentrations cannot be considered in exposure analyses based on emission estimates alone. Assessments based on ambient monitoring data provide a more complete accounting of actual risk due to formaldehyde, because they cover concentrations resulting from both emissions and atmospheric formation. The NESHAP estimate based on emissions was 1.6 per year; the ambient data resulted in an estimate of 191 per year.

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[14] Zaragoza, L. "Calculating Effects of Carbon Tetrachloride and Other Chlorocarbons on Increases in Skin Cancer from Stratospheric Ozone Depletion," EPA, OAQPS Draft. July 25, 1984.

4. 35-County Study

Background

In contrast to the national scope of the NESHAP study, the 35-County Study was designed to address the air toxics problem from a more local perspective.[15] Building on the work of EPA's Integrated Environmental Management Division (IEMD) in its geographic demonstration projects in Philadelphia, Baltimore and Santa Clara Valley, this analysis explored:

- the incidence of cancer resulting from exposure to several pollutants and sources in specific localities;
- the pollutants and sources that are the most significant contributors to incidence; and,
- the geographic variability of pollutants, sources, and exposures.

The analysis focused on traditional sources, i.e., large point sources such as power plant and industrial facilities, and area sources, such as motor vehicles, space heating, gasoline marketing, and solvent usage. However, it also included "nontraditional" sources, such as wood stoves, waste oil combustion, and sewage treatment plants. Because of data limitations we could not make emission estimates or perform any extensive exposure modeling for TSDFs (hazardous waste treatment, storage and disposal facilities), Superfund sites, hazardous waste in boilers, municipal waste incinerators, municipal landfills, and sewage sludge incinerators. The Agency has initiated various studies to explore emissions and risks for most of these sources in more detail. Information on these efforts, as well as any preliminary findings, is provided in the section on Other Sources, Pollutants and Pathways at the end of this chapter.

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[15] Versar; American Management Systems, Inc., "Hazardous Air Pollutants: An Exposure and Risk Assessment for 35 Counties," U.S. EPA Contract #68-01-6715, September 1984.

The analysis characterized exposure and risk associated with 22 compounds (see Table 2). Most of these compounds were screened using one or more of the following criteria:

- Sufficient evidence of carcinogenicity;
- Significant release rates; and
- Readily available emissions information.

Emission estimates for routine emissions were developed using several techniques. Whenever possible, the analysis relied on plant-specific data and EPA documents on emissions from specific source categories. Where this information was unavailable, surrogate loadings were developed using the information in the National Emissions Data System (NEDS), and apportioning factors that speciate the volatile organic compound and particulate matter data into individual toxic constituents. NEDS data vary a great deal in quality, and some of the data are very poor. However, an extensive effort was made to screen NEDS data for the 35 counties to correct for any obvious inaccuracies in release rates, source locations and stack specifications.

We developed special algorithms for the following sources: POTWs, waste oil combustion, woodsmoke, and gasoline marketing. To calculate releases for selected volatile compounds from sewage treatment plants, we modeled thirteen prototype POTWs using information provided by EPA's Industrial Facilities Discharge (IFD) file, the NEDS survey, and a study conducted by the effluent guidelines program

TABLE 2

35-COUNTY STUDY: PRELIMINARY APPROXIMATION OF ANNUAL INCIDENCE

Pollutants Having Some Evidence of Carcinogenicity*	Preliminary Approximation of Incidence** (20% of U.S. Population)
PIC***	124.3
Benzene	18.5
Chromium†	13.4
Formaldehyde	10.0
Vinyl chloride	8.2
Trichloroethylene	6.8
Gasoline Vapors	6.8
Perchloroethylene	6.7
Acrylonitrile	4.2
Coke oven emissions	2.4
Ethylene dichloride	1.5
Arsenic	1.1
Cadmium	1.1
Benzo(a)pyrene	1.1
Ethylene dibromide	1.0

* The weight of evidence of carcinogenicity for the compounds listed varies greatly, from very limited to very substantial. Further, the extent of evaluation and health review performed varies considerably among compounds. However, for the purposes of this report, a conservative scenario (i.e., that all compounds examined could be human carcinogens) has been assumed.

** Because of the uncertainties in the data used to make these estimates, they should be regarded as rough approximations of total incidence. Estimates for individual compounds are much less certain. These incidence estimates have been performed to provide a rough idea of the possible total magnitude of the air toxics problem, and will be used only for priority-setting and to provide policy guidance.

*** "Products of Incomplete Combustion" (PIC) refers to a large number of compounds, probably consisting primarily of polynuclear organics. The PIC unit risk value was derived from dose-response data which use B(a)P levels as a surrogate for PIC or total air pollution. There are many limitations of using the B(a)P surrogate method to estimate PIC risks: all PIC estimates presented in this report must be regarded as highly uncertain. Refer to pp. 20-25 for a more detailed explanation of how the PIC unit risk value was derived.

† Risk estimates assume that all species of chromium and nickel are carcinogenic, although only certain species have evidence of carcinogenicity. Current data do not allow differentiation among species.

TABLE 2 (Cont.)

35-COUNTY STUDY: PRELIMINARY APPROXIMATION OF ANNUAL INCIDENCE

Pollutants Having Some Evidence of Carcinogenicity*	Preliminary Approximation of Incidence** (20% of U.S. Population)
Carbon tetrachloride	0.2
Chloroform	0.1
Styrene	0.02
Beryllium	0.01
1,3-Butadiene	0.01
Pentachlorophenol	≤ 0.01
Total	207.4

* The weight of evidence of carcinogenicity for the compounds listed varies greatly, from very limited to very substantial. Further, the extent of evaluation and health review performed varies considerably among compounds. However, for the purposes of this report, a conservative scenario (i.e., that all compounds examined could be human carcinogens) has been assumed.

** Because of the uncertainties in the data used to make these estimates, they should be regarded as rough approximations of total incidence. Estimates for individual compounds are much less certain. These incidence estimates have been performed to provide a rough idea of the possible total magnitude of the air toxics problem, and will be used only for priority-setting and to provide policy guidance.

to determine the fate of priority pollutants in 50 POTWS.[16,17] The sewage treatment plants in each of the 35 counties were assigned to one of the model plants based on the following factors: the percent of inflow to the POTW attributable to industrial dischargers; the types of industries that discharge to the POTW; and the level of treatment at the POTW. The sewage treatment plant emissions were modeled as point sources in the exposure assessment.

Toxic emissions from waste oil combustion were characterized using data from EPA Office of Solid Waste (OSW) documents on: the typical contaminant concentrations found in used oil; the estimated amount of waste oil burned in each state; the destruction efficiencies for metals and organic compounds burned in industrial and in residential, institutional and commercial boilers; and the percentage of total waste oil burned in each type of boiler.[18] The study of waste oil focused on chromium, cadmium, beryllium, arsenic, benzene, benzo(a)pyrene, perchloroethylene, and trichloroethylene. Waste oil emissions were modeled as area sources.

Air toxics releases from woodsmoke were estimated for two sources--fireplaces and wood stoves.[19] Using available information,

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[16] Fate of Priority Pollutants in Publicly Owned Treatment Works, Vol. I., (EPA 440/1-82-303), September 1982.

[17] For further explanation on the method for estimating POTW volatilization, see Versar/American Management Systems, Inc., "Hazardous Air Pollutants: An Exposure and Risk Assessment for 35 Counties," Appendix F-2, September 1984.

[18] For further explanation on the method for estimating toxics emissions from waste oil combustion, see Versar American Management Systems, Inc. "Hazardous Air Pollutants," Appendix C.

[19] For further information on the method for estimating woodsmoke emissions, see Versar/American Management Systems, Inc., "Hazardous Air Pollutants," Appendix B.

we developed factors for five compounds (benzo(a)pyrene, formaldehyde, beryllium, cadmium, and arsenic) relating pollutant emissions to the quantity of wood burned in each county. Data on wood consumption in each county were obtained from NEDS, and the breakdown on the amount of wood burned in woodstoves vs. fireplaces in each area was provided by an industry association. We modeled wood smoke as an area source.

Finally, air toxics emissions from gasoline marketing were calculated using volatile organic compound data in NEDS and apportioning factor developed from varied sources. The pollutants considered were: gasoline vapors, benzene, ethylene dibromide, and ethylene dichloride.

As to the choice of geographic sites, we decided to concentrate on counties, as data are rarely disaggregated below this level. We chose 35 counties to explore in detail, and each county fell into one of three categories:

- densely populated, highly industrialized;
- densely populated, low industrial activity; or
- low population density, highly industrialized.

The counties were chosen to represent a wide range of industrial bases and geographic locations. Although they contain only about one percent of the counties in the U.S., the 35 counties account for roughly 20 percent of U.S. population (1980 Census Data), 20 percent of total national VOC emissions, and 10 percent of total PM emissions.

As with the NESHAP analysis and other Agency studies on exposure, the 35-County Study employed dispersion modeling to calculate dose and exposure. EPA's Office of Toxic Substances' fate and transport model, GAMS, was used in this effort. To facilitate running the model more quickly and efficiently, we used an approach that only allowed us to calculate annual aggregate incidence for the 35 counties.

Findings

Multiplying the results from the exposure modeling by the appropriate unit risk values (Attachment A) resulted in the incidence estimates presented in Table 2. The estimated aggregate incidence of cancer for the 22 pollutants and 35 counties is 207 per year. As shown, 8 substances account for roughly 95 percent of the total risk. These pollutants, ranked in descending order, are as follows: PIC; benzene; chromium; formaldehyde; vinyl chloride; trichloroethylene; gasoline vapors; and perchloroethylene. PIC alone contributes 60 percent to total incidence.

Many of the basic problems discussed in the NESHAP analyses are applicable to the 35-County Study (see pp. 28-33). Also, the 35-County Study considered emissions of carbon tetrachloride from only a limited number of sources. Background concentrations due to the long half-life of carbon tetrachloride were not modeled, although they may significantly contribute to cancer risks.

5. Ambient Air Quality Study

Background

As part of the overall study, we used ambient air quality data to estimate cancer incidence and individual risks.[20] Two basic groupings of compounds were used in this analysis: those for which fairly extensive data were available--four metals and B(a)P--and those for which less extensive data could be found--nine organic compounds. The metals and B(a)P data were drawn from the National Air Data Bank's Storage and Retrieval of Aerometric Data (SAROAD) system. In contrast, the data for organic compounds came from a variety of sources, principally from studies which used diverse sampling and analytical methods and sampling periods.

Every attempt was made to gather all available ambient data on air toxics. For example, for organic compounds the data base incorporated data compiled from a variety of sources by Dr. Hanwant Singh of SRI International and from more recent monitoring studies performed in Baltimore, Los Angeles, Houston, Philadelphia, and in northern New Jersey. As far as we know, this effort represents the most comprehensive attempt yet to compile nationwide data for toxic air pollutant and to perform risk assessments based on those data.

It is appealing to use ambient air quality data--as opposed to modeled estimates--to estimate risks because these data represent the actual ambient concentrations to which people are exposed.

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[20] Hunt, Bill, et al., "Estimated Cancer Incidence Rates from Selected Toxic Air Pollutants Using Ambient Air Data," U.S. EPA, revised March 1985.

However, the reader is reminded of three cautions which have been discussed previously. First, we must assume that data collected at a limited number of sites can be extrapolated to represent city-wide and county-wide levels, and that these data in turn can be extrapolated to the national level. Second, we must often use data collected over a short time period (e.g., 24 hours,) and assume that in the aggregate they are representative of longer term concentrations (e.g., annual averages). Finally, we assume that people are continuously exposed to levels equal to those of ambient air.

National estimates of cancer incidence were calculated for metals (see Table 3) by estimating county averages based on 1979-1982 data for the approximately 170 counties that had data, by using these averages to extrapolate to those counties that lacked data, and then by applying the unit risk values presented in Attachment A. We estimated a national incidence for PIC by dividing the country into 11 regions and using urban/rural B(a)P concentrations in combination with urban/rural population figures for each region.

Estimating incidence for the volatile organic compounds was somewhat more difficult, given that ambient data on these compounds are scarce and often derived from short-term studies. To provide at least minimal seasonal balance when computing annual averages, we established a data completeness criterion[21] for organic compounds in urban areas. This greatly reduced the amount of data that could be used. Only data from studies performed in

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[21] More than two sites per county, and at least ten samples over two quarters in a single calender year.

TABLE 3

AMBIENT AIR QUALITY STUDY: PRELIMINARY APPROXIMATION OF ANNUAL INCIDENCE

Pollutants Having Some Evidence of Carcinogenicity*	Preliminary Approximation of Incidence**	Incidence per Million Population**
Arsenic	60	0.26
Benzo(a)pyrene	5	0.02
PIC***	610	2.65
Benzene	234	1.02
Beryllium	<1	<0.01
Cadmium	15	0.06
Carbon tetrachloride	43	0.19
Chloroform	17	0.07

* The weight of evidence of carcinogenicity for the compounds listed varies greatly, from very limited to very substantial. Further, the extent of evaluation and health review performed varies considerably among compounds. However, for the purpose of this report, a conservative scenario (i.e., that all compounds examined could be human carcinogens) has been assumed.

** Because of the uncertainties in the data used to make these estimates, they should be regarded as rough approximations of total incidence. Estimates for individual compounds are much less certain. These incidence estimates have been performed to provide a rough idea of the possible total magnitude of the air toxics problem, and will be used only for priority-setting and to provide policy guidance.

*** "Products of Incomplete Combustion" (PIC) refers to a large number of compounds, probably consisting primarily of polynuclear organics. The PIC unit risk value was derived from dose-response data which use B(a)P levels as a surrogate for PIC or total air polluton. There are many limitations of using the B(a)P surrogate method to estimate PIC risks: all PIC estimates presented in this report must be regarded as highly uncertain. Refer to pp. 20-25 for a more detailed explanation of how the PIC unit risk value was derived.

TABLE 3 (Cont.)

AMBIENT AIR QUALITY STUDY: PRELIMINARY APPROXIMATION OF ANNUAL INCIDENCE

Pollutants Having Some Evidence of Carcinogenicity*	Preliminary Approximation of Incidence**	Incidence per Million Population**
Chromium†	242	1.05
Ethylene dichloride	11	0.05
Formaldehyde	191	0.83
Methyl chloride	1	<0.01
Methylene chloride	1	<0.01
Perchloroethylene	22	0.10
Trichloroethylene	18	0.08
Vinylidene chloride	62	0.27
Total	1532	6.7

* The weight of evidence of carcinogenicity for the compounds listed varies greatly, from very limited to very substantial. Further, the extent of evaluation and health review performed varies considerably among compounds. However, for the purpose of this report, a conservative scenario (i.e., that all compounds examined could be human carcinogens) has been assumed.

** Because of the uncertainties in the data used to make these estimates, they should be regarded as rough approximations of total incidence. Estimates for individual compounds are much less certain. These incidence estimates have been performed to provide a rough idea of the possible total magnitude of the air toxics problem, and will be used only for priority-setting and to provide policy guidance.

† Risk estimates assume that all species of chromium are carcinogenic, although only certain species have evidence of carcinogenicity. Current data do not allow differentiation among species.

Baltimore, Philadelphia, Los Angeles, Houston, and northern New Jersey met the criterion. For these cities, an average level was calculated for each organic compound, and these averages were then combined with population figures to calculate incidence. Next, these estimates were extrapolated to the national level by using urban population data. Nonurban risks were calculated by using nonurban pollutant levels and population data, and these were added to urban risks to obtain national estimates.

Findings

As Table 3 shows, seven compounds are associated with greater than 50 cancers per year: arsenic, PIC, benzene, chromium, formaldehyde, and vinylidene chloride. The national incidence estimate based on ambient data for the compounds shown in Table 3 is approximately 1,530 per year. The estimated incidence per million population for those pollutants is about 6.7 per year.

Individual lifetime risks were also estimated for metals, PIC, and organics (Table 4). Individual risks ranged up to 10^{-3} for some of the trace metals and PIC, whereas individual risks for the organics tended to be in the range of 10^{-4} and lower. It should be noted that the sites where these data were collected are generally not located near points of expected maximum concentrations. Therefore, the individual risk estimates for single pollutants based on air quality data tended to be lower than those based on exposure modeling of emissions from point sources.

However, to provide a better understanding of risks in urban areas, we estimated individual risks not only on an individual pollutant basis, but also for many pollutants measured at the same site. The results of this analysis are presented in

Table 5 for several urban areas in which extensive ambient monitoring has been performed. These multipollutant individual risks represent the summed individual risks at each site using monitoring data that were available for 10 to 15 organics, metals, and PIC. Table 5 shows that these multipollutant individual lifetime risks range around 1 X 10^{-3} for those areas with sufficient data for analysis. Lifetime individual risks for single pollutants at these sites varied from 10^{-3} to 10^{-9}; pollutants causing risks in the 10^{-3} to 10^{-4} range included chromium, PIC, benzene, and formaldehyde. To our knowledge, none of the monitoring sites were near major point sources of the relevant compounds, although all sites were centrally located in major urban areas.

It is important to note that the uncertainties associated with extrapolating data collected at a few monitoring sites to an entire urban area do not apply to these estimates of multipollutant individual risk. All that is involved is summing individual risks from a pollutant mixture at a given urban location. Thus, with the assumption that risks are additive, we can say that, even in neighborhoods not located near major point sources, urban dwellers may experience individual risks of 10^{-3} to 10^{-4} due to multi-pollutant air exposures.

TABLE 4

AMBIENT AIR QUALITY STUDY: PRELIMINARY APPROXIMATION OF INDIVIDUAL LIFETIME RISKS

Pollutants Having Some Evidence of Carcinogenicity*	Preliminary Approximation of Maximum Lifetime Individual Risk**
Arsenic	3.99×10^{-3}
B(a)P	2.47×10^{-5}
PIC***	3.15×10^{-3}
Benzene	1.54×10^{-4}
Beryllium	2.40×10^{-7}
Cadmium	1.47×10^{-3}
Carbon tetrachloride	1.54×10^{-4}
Chloroform	7.70×10^{-5}

* The weight of evidence of carcinogenicity for the compounds listed varies greatly, from very limited to very substantial. Further, the extent of evaluation and health review performed varies considerably among compounds. However, for the purpose of this report, a conservative scenario (i.e., that all compounds examined could be human carcinogens) has been assumed.

** Because of the uncertainties in the data used to make these estimates, they should be regarded as rough approximations of maximum lifetime individual risk. Estimates for individual compounds are very uncertain. These risk estimates have been performed to provide a rough idea of the possible total magnitude of the air toxics problem, and will be used only for priority-setting and to provide policy guidance.

*** "Products of Incomplete Combustion" (PIC) refers to a large number of compounds, probably consisting primarily of polynuclear organics. The PIC unit risk value was derived from dose-response data which use B(a)P levels as a surrogate for PIC or total air polluton. There are many limitations of using the B(a)P surrogate method to estimate PIC risks: all PIC estimates presented in this report must be regarded as highly uncertain. Refer to pp. 20-25 for a more detailed explanation of how the PIC unit risk value was derived.

TABLE 4 (Cont.)

AMBIENT AIR QUALITY STUDY: PRELIMINARY APPROXIMATION OF INDIVIDUAL LIFETIME RISKS

Pollutants Having Some Evidence of Carcinogenicity*	Preliminary Approximation of Maximum Lifetime Individual Risk**
Chromium†	1.44×10^{-3}
Formaldehyde	4.91×10^{-5}
Methyl chloride	4.60×10^{-7}
Methylene chloride	8.28×10^{-7}
Perchloroethylene	1.88×10^{-5}
Trichloroethylene	2.59×10^{-5}
Vinylidene chloride	8.06×10^{-5}

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* The weight of evidence of carcinogenicity for the compounds listed varies greatly, from very limited to very substantial. Further, the extent of evaluation and health review performed varies considerably among compounds. However, for the purpose of this report, a conservative scenario (i.e., that all compounds examined could be human carcinogens) has been assumed.

** Because of the uncertainties in the data used to make these estimates, they should be regarded as rough approximations of maximum lifetime individual risk. Estimates for individual compounds are very uncertain. These risk estimates have been performed to provide a rough idea of the possible total magnitude of the air toxics problem, and will be used only for priority-setting and to provide policy guidance.

† Risk estimates assume that all species of chromium are carcinogenic, although only certain species have evidence of carcinogenicity. Current data do not allow differentiation among species.

TABLE 5

AMBIENT AIR QUALITY STUDY: PRELIMINARY APPROXIMATION OF ADDITIVE LIFETIME RISKS*

Urban Area A	Monitoring Site 1	2.3×10^{-3}
	Monitoring Site 2	2.3×10^{-3}
Urban Area B	Monitoring Site 1	0.7×10^{-3}
	Monitoring Site 2	0.7×10^{-3}
Urban Area C	Monitoring Site 1	1.1×10^{-3}
	Monitoring Site 2	1.0×10^{-3}
Urban Area D	Monitoring Site 1	0.8×10^{-3}
	Monitoring Site 2	0.8×10^{-3}

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* These estimates are based on a sum of estimated lifetime individual risks for PIC (products of incomplete combustion), two to three metals and six to ten organic compounds for each monitoring site. Because of the uncertainties in the data used to make these estimates, they should be regarded as rough approximations of individual risk. Estimates for individual compounds are much less certain. These incidence estimates have been performed to provide a rough idea of the possible total magnitude of the air toxics problem, and will be used only for priority-setting and to provide policy guidance.

6. Other Pollutants, Sources and Pathways

One of the principal findings of this study of air toxics is that there are important gaps in our knowledge of this problem. This study estimates cancer risks caused by 15-45 substances, when there may be many more potential carcinogens in the ambient air. The International Agency for Research on Cancer (IARC), the National Toxicology Program, and EPA's Carcinogen Assessment Group have each identified over 100 compounds as carcinogenic. Many of these compounds are probably not air pollutants, but it is clear that this study does not quantitatively address a large number of pollutants that exist in significant quantities in the ambient air. This study attempted also to address all known or suspected sources of air toxics, as well as known pollutants. Unfortunately, we were unable to quantify the risks caused by several source categories, including several nontraditional sources. In addition, each of the individual analyses missed some sources or pollutants.

However, some of the sources and pollutants not included in the major analyses have been subjects of quantitative analysis by others. The following section summarizes available information on the pollutants and sources that (1) were not covered by the individual analyses; or (2) could not be quantitatively assessed because of data limitations. As with most of the analyses these represent the situation early in 1985 and changes are inevitable over time.

POLLUTANTS

Dioxin

Only isolated estimates are provided for individual risks from emissions of dioxin, and these are limited to municipal incinerators. The exposure pattern for dioxin appears to be complex and available data are inconsistent. However, this is true for many compounds that we have included in the study. Dioxin is unique because exposure and risk are being studied in great detail by EPA's Dioxin Task Force. The study team believed that there was little value at this time in attempting an estimate of the aggregate risk from air exposure for a pollutant that is currently being analyzed elsewhere in such detail.

Asbestos

EPA's Office of Pesticides and Toxic Substances is actively considering regulations for asbestos, since past use of asbestos-containing building materials can lead to indoor contamination. Asbestos is also commonly found in the ambient air, although at much lower levels than indoors, and selected sources are already covered by federal emission standards under Section 112 of the Clean Air Act. Sampling and analysis for asbestos in the atmosphere presents significant problems and concentrations vary by several orders of magnitude. The available data suggest an average of three nanograms/m^3 and 30 fibers per nanogram.[22] Coupling this with an average risk factor for lung cancer and mesothelioma,[23] gives a national estimate of over 100 excess cancers per year, or about 0.5 per million population per year. This estimate covers outdoor exposures only.

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[22] "Guidance for Controlling Friable Asbestos-Containing Materials in Buildings." U.S. EPA Office of Pesticides and Toxic Substances, EPA 560/5-83-002, March 1983.

Radionuclides

EPA's Office of Radiation Programs (ORP) has evaluated radionuclides as a hazardous pollutant, based on the widespread human exposure to radionuclides in the ambient air, and on numerous studies that document the incidence of cancer resulting from exposure to ionizing radiation in many species of animals and human populations. ORP has recently summarized its exposure and risk assessment for radionuclides.[24] As shown in Table 6, the total national estimated incidence for radionuclides is 16 per year; maximum lifetime individual risks range from 5×10^{-2} to 4×10^{-7}. The major sources of radionuclides include nuclear power plants, national defense weapon facilities, industrial plants, coal-fired boilers and natural sources. The incidence calculation does not consider exposure to indoor concentrations of radon.

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[23] Schneiderman, Nisbet, and Brett, "Assessment of Risks Posed by Exposure to Low Levels of Asbestos in the General Environment," Berichte, Bundes-Gesundheits-AMT, pp. 3-1 to 3-28, April 1981.

[24] Hardin, J. "Issue Paper. National Air Toxics Problem: Radionuclides." EPA, Office of Radiation Programs, August 1984. Update provided verbally on January 31, 1985.

TABLE 6

ESTIMATES OF INCIDENCE AND INDIVIDUAL RISK DUE TO RADIONUCLIDES EMITTED TO AIR*

Source	Annual Cancer Incidence	Maximum Individual Lifetime Risk
Dept. of Energy Facilities	0.07	8×10^{-4}
Nuclear Regulatory Commission (NRC) Licensed Facilities	<0.01	2×10^{-5}
Federal Facilities	<0.01	4×10^{-7}
Uranium Fuel Cycle Facilities	5	1×10^{-4}
Uranium Mill Tailings Piles	7	4×10^{-2}
Uranium Mines	1.0	5×10^{-2}
Phosphorus Plants	0.06	1×10^{-3}
Coal-Fired Boilers	3	3×10^{-5}
Sources of Natural Radionuclides to Air	<0.1	2×10^{-4}
TOTAL	16	

* Because of uncertainties in underlying data, the values presented in this table should be regarded as estimates of incidence and maximum lifetime risk. This table was provided by EPA's Office of Radiation Programs. Please refer to footnote 24 for a more detailed explanation of the methodology.

Recent studies have indicated that indoor air concentrations of various pollutants can greatly exceed ambient conditions. As a result, risk assessments based on ambient levels may understate the actual risk. For radionuclides, recent estimates place the annual incidence of cancer due to indoor radon exposure at between 1,000 and 20,000. A more detailed discussion of the ramifications of indoor air on the hazardous air pollutant problem is provided in the section of this report on Perspective and Context.

Other Pollutants

It is apparent that urban ambient air is characterized by the presence of hundreds of organic compounds; fine particulate matter, including metals and organic particulates; and significant concentrations of the other criteria pollutants, including sulfur and nitrogen oxides, and carbon monoxide. Relatively few data are available on how all of these substances may interact once they enter the human body.

An example of the complexity of urban air is shown in Figure I, a gas chromatogram from EPA's Integrated Environmental Management Division's monitoring program in a major metropolitan area. It represents the concentrations and number of gaseous organics in the ambient air as detected by gas chromatography/mass spectroscopy. Each peak represents a separate organic compound. The peaks corresponding to some compounds are labeled. Almost 50 tentatively identified compounds added up to the following totals:

Figure I

DRAFT

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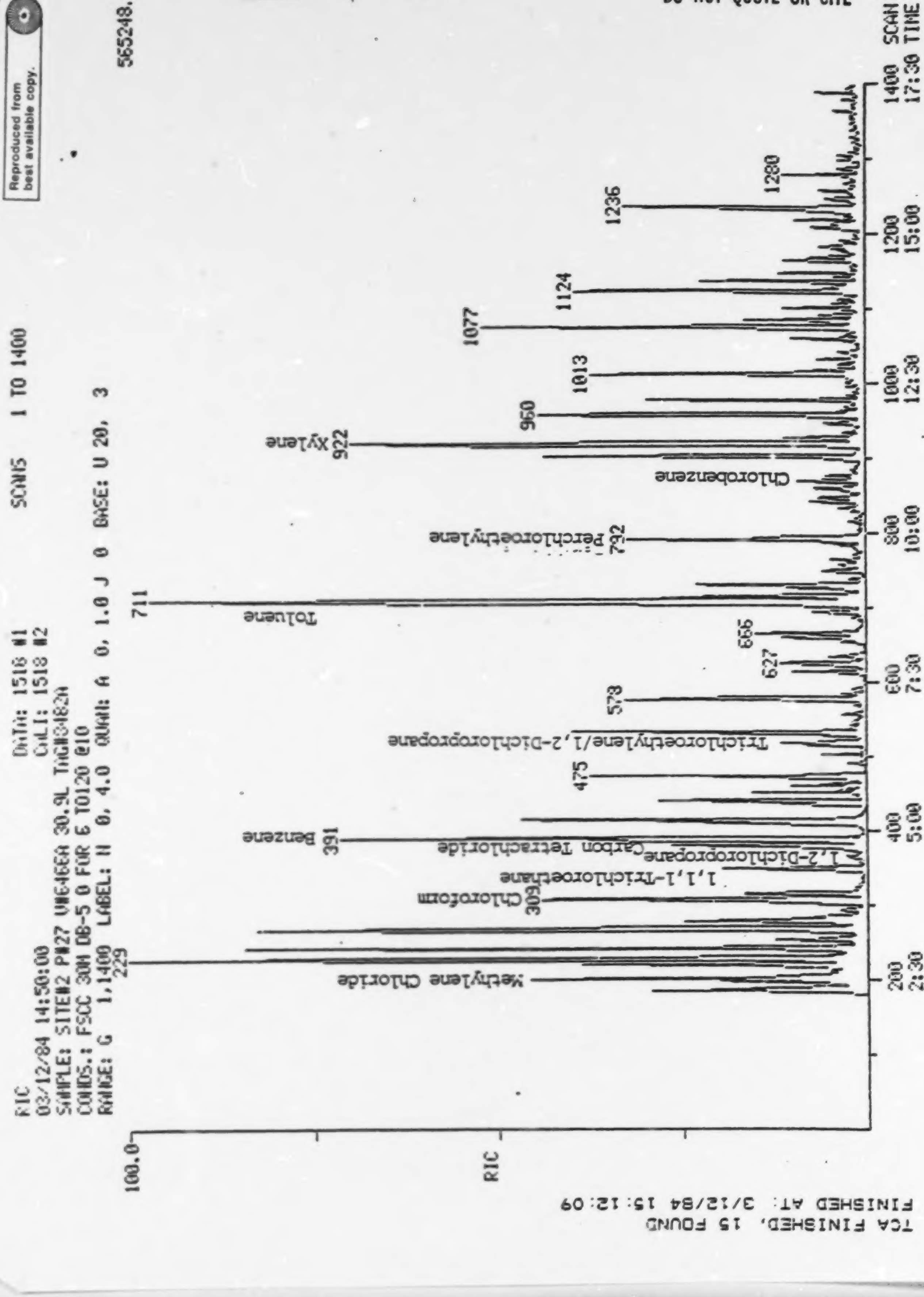

Alkanes	39.1 ug/m3
Aromatics	34.8 ug/m3
Halogenated compounds	9.8 ug/m3
Oxygenated compounds	7.5 ug/m3
Alkenes	3.4 ug/m3

SOURCES

Atmospheric Transformation

Most population exposure models begin with estimates of emissions, and thus inherently cannot handle toxic compounds that may be formed or rapidly destroyed in the atmosphere. The exposure models used in the NESHAP and 35-County studies assume that all exposures occur within several hours of emission (within 20 km to 50 km of the source) and no corrections are made for transformation of pollutants in the atmosphere.

As part of the study, EPA's Office of Research and Development was asked to review the possibility that chemical reactions in the atmosphere could form toxic compounds or increase the potency of emitted pollutants.[25] Ozone is the prime example of this phenomenon for criteria pollutants. Although work in this area has not been extensive, the study identified several potentially significant examples of atmospheric transformation. A few of the situations discussed in the review are summarized below.

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[25] Bufalini, J., B. Gay and B. Dimitriades, "Production of Hazardous Pollutants Through Atmospheric Transformation," U.S. EPA Office of Research and Development, June 1984.

Formaldehyde and acrolein are formed readily in a variety of photochemical reactions involving emissions from many types of natural and man-made hydrocarbon emissions. For formaldehyde, an important contributor to total risk in this study, atmospheric formation may produce several times the amount directly emitted from all sources. This may explain some of the major differences between the risks estimates obtained by using exposure models versus measured data.

Experimental evidence is also available that photooxidation of compounds with little evidence of carcinogenicity, such as toluene and propylene, produce substances with significant mutagenicity. The compounds responsible have not been fully identified. In other experiments, phosgene has been produced photochemically from chlorinated hydrocarbons, such as solvents. The studies suggest that a hundred times more phosgene may be formed in the atmosphere than is emitted directly.

As a final example, studies of the mutagenic activity of polycyclic organic particulates show large increases in activity when the material is subjected to mixtures of ozone and nitrogen oxides. Organic particulates are a ubiquitous group of pollutants generally associated with incomplete combustion (mobile sources, small units burning wood, coal, and oil). They are represented by PIC in this report and may be a major contributor to risks from air toxics in many communities.

Gasoline Marketing

Gasoline marketing includes a series of emission points ranging from major bulk terminals to filling of individual vehicles at self-service stations. These sources are receiving special attention within EPA because of the significance of their emissions, the potential economic impact of control on thousands of service stations, the alternative of onboard controls, litigation on benzene under Section 112, and the importance of gasoline marketing for ozone attainment strategies. EPA's Gasoline Marketing Task Force has developed detailed estimates of the risk from these facilities that cover benzene, ethylene dibromide, ethylene dichloride, and gasoline vapors. The Task Force estimated an aggregate incidence of 43 excess cancers per year from all gasoline marketing sources. This estimate was used in portions of this study.

Woodstoves

As indicated in the Ambient Air Quality and 35-County studies, products of incomplete combustion may be a significant hazardous air pollutant problem. Recent studies suggest that residential wood combustion contributes about 40 percent of total national emissions of polycyclic organic matter (POM). POM compounds found in wood smoke include BaP and polycyclic organic ketones. In addition, one EPA study suggests that the emission rate of mutagenic and carcinogenic substances from woodstoves is at least several orders of magnitude greater than from other combustion sources used to heat homes. The results of the 35-County Study supported this concern: roughly 80 percent of the annual estimated cancer incidence for BaP from heating in the 35 counties was attributable to wood combustion.

There are currently no data on the human health risks attributable specifically to wood smoke. As a result, the 35-County Study assessed the potential human health hazard posed by wood combustion, considering the health effects associated with only a few individual compounds (BaP, formaldehyde, cadmium, beryllium, and arsenic). The estimated annual cancer incidence in the 35 counties resulting from exposure to these compounds is 27, including the use of BaP exposure as a surrogate for PIC. However, it should be noted that the 35 counties analyzed are not representative of those areas where wood combustion is of greatest concern, such as parts of New England, Montana, and Colorado. Thus, the estimates for woodstoves understate risks for such areas.

EPA has established a committee that soon will recommend research and regulatory initiatives for woodstoves to the Agency. These recommendations will include: a comprehensive research program on health effects, emission testing procedures, and control techniques; establishment of a variety of technical assistance programs on wood smoke; and consideration of a new source performance standard for woodstoves. The Integrated Cancer Assessment Project, which is scheduled to begin this fall, also plans to assess the contribution of woodstove emissions to the total organics, POM, and mutagenic activity in the airsheds to be studied.

Sewage Treatment Plants

Sewage treatment plants have become a source of interest for air releases primarily because of work undertaken by EPA's Integrated Environmental Management Division (IEMD). Preliminary findings suggest that many Publicly Owned Treatment Works (POTW's) located in urban areas with indirect industrial dischargers may emit volatile organic compounds in excess of 100 kkg/year. Using a POTW algorithm developed for the 35-County Study, we estimated an annual cancer incidence in the 35 counties of 2.3 for the nine pollutants that we were able to consider.

Given the paucity of data on air releases from sewage treatment plants, there is a need to explore this area in more detail. The IEMD will continue to monitor and model POTW's as part of its activities in future work on geographic sites; EPA's Pretreatment Task Force may also explore potential air emissions from sewage treatment plants.

Hazardous Waste Combustion in Boilers

Although insufficient data were available to quantify the problem of disposal of hazardous waste in boilers, EPA's Office of Solid Waste (OSW) has attempted to assess the risk resulting from the burning of hazardous waste using a model boiler approach. This study considered three boiler sizes and estimated exposure and risk for three metropolitan areas: New York, Cleveland, and Los Angeles. These cities were chosen because they represent a wide variety of exposure characteristics for densely populated, highly industrialized areas. As information on the quantity, distribution, and toxic content of the hazardous material burned was limited at the time OSW initiated this analysis, this study tends to depict a worst-case scenario. The study findings suggest that:

- Lifetime individual risks for the most exposed individual in these three regions range from 5×10^{-6} to 1.4×10^{-5}, depending on the boiler type.[26]

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[26] "Draft Preliminary Risk Assessment for Burning Hazardous Waste in Boilers." Office of Solid Waste, EPA. February 16, 1984, p. 2.

- Lifetime risks to the average exposed individual in these three regions ranged from 1.2×10^{-7} to 6×10^{-7}, depending on the boiler type.
- Estimated annual cancer incidence in these three regions ranges from 0.01 to 0.2, depending on the boiler type.
- The risk associated with metals is potentially much higher than that for organics. Using metal concentrations found in virgin fuel, the analysis shows that metals contribute roughly 52 percent to the total estimated incidence. Burning hazardous material with metal concentrations higher than these could increase the problem.

OSW has also just completed the Survey of Handlers and Burners of Used or Waste Oil and Waste-Derived Fuel Materials (Track II) which should provide useful information for future studies on risk. Although OSW has only begun to analyze the survey results, some preliminary findings on the burning of waste-derived fuel material (WDFM)[27] are as follows:

- 924 million gallons of WDFM are burned each year;
- About 200 million gallons of this material are estimated to be hazardous, as defined by the Resource Conservation and Recovery Act (RCRA); and,
- Chemical manufacturing, pulp and paper, lumber, primary metals, and petroleum refining industries burn about 90% of total WDFM.

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[27] "Status of the Data Collection Effort for the U.S. EPA: Survey of Handlers and Burners of Used or Waste Oil and Waste-Derived Fuel Material: Track II." December 1983, pp. 3-4. It should be noted that WDFM is a broader category than hazardous waste. For the purposes of the survey, WDFM was defined as "any material that is a constituent of a fuel, or is destined to be burned as a fuel, that is not a conventional fuel material." Examples of conventional fuel are: distillate fuel oil; residual fuel oil; natural gas; coal; liquified petroleum gas; and refuse-derived fuels.

OSW is initiating analyses to identify boiler operating practices, to characterize the specific wastes being burned, and to determine the quantity and geographic distribution of these hazardous wastes. This information will be used to complete an exposure and risk assessment that will support the Regulatory Impact Analysis for the regulation of burning hazardous waste and used oil fuels. The tentative schedule for completing this analysis is September 1985.

Waste Oil Combustion

The Office of Solid Waste (OSW) estimates that 500 to 550 million gallons of used oil are recycled as fuels each year.[28] While most of these fuels are burned in boilers, they may also be burned in kilns, space heaters, and diesel engines. Because of contamination during use and because of mixing, used oils typically contain elevated levels of toxic metals--such as arsenic, cadmium, and chromium--and organics, such as BaP and PCBs. Burning used oils may result in elevated ambient concentrations of some of these contaminants, particularly when the combustion sources are clustered.[29] The potential emissions of metals appear to contribute more to risk than organic emissions. The 35-County Study corroborated the importance of metals: of a total annual cancer incidence of 6.7 from waste oil combustion in the 35 counties, 90 percent was accounted for by chromium and 9.5 percent by arsenic.

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[28] U.S.EPA, "Composition and Management of Used Oil Generated in the U.S." December 1983.

[29] U.S.EPA, "A Risk Assessment of Waste Oil Burning in Boilers and Space Heaters." Draft, January 1984.

OSW is currently developing emission standards for waste oil combustion and will evaluate these risks more closely, for a variety of exposure pathways.

Operational Hazardous Waste Facilities

Over the past several years, there has been an increasing concern that operational treatment, storage, and disposal facilities (TSDF's) for hazardous wastes may be an important source of air emissions. There have been many efforts to quantify releases of volatile organic compounds from TSDF's. In general, these analyses have either focused on individual facilities, using ambient monitoring to estimate atmospheric pollutant concentrations, or on national estimates, employing emission models to assess air releases. In addition, Westat, Inc. recently completed an extensive survey for the Office of Solid Waste (OSW) that provides a great deal of background information on the quantity, constituency, and distribution of hazardous waste generated and managed by TSDF's throughout the country. The survey estimates that a total of 71.3×10^9 gallons (264×10^6 metric tons) of waste are managed by hazardous waste facilities, and that over 50 percent of this quantity is treated, stored, and/or disposed of in impoundments and landfills. In addition, the survey indicates that the chemical industry generates over 70 percent of total hazardous waste. If we assume that a substantial amount of the chemical industry's waste consists of volatile organic compounds, there is a clear potential for significant air emissions from TSDF's.

Although the survey yields some interesting findings on the types and quantity of hazardous waste managed at TSDF's, it is nonetheless one step removed from actual emission estimates. There have been several recent attempts to estimate releases from TSDF's at the national level using emission modeling. Unfortunately, these studies have been severely criticized. It is apparent that estimating volatilization from TSDF's is still in its infancy, and that these models generally require further refinement and validation.

The monitoring data on ambient concentrations around specific TSDF's are probably more persuasive in making the case that TSDF's are potentially significant sources of air toxics. To explore the potential hazard from the volatilization of organic compounds, we used air toxics concentration data from a study of one TSDF, the BKK landfill in California.[30] This was the only data set found that attempted to measure actual ambient concentrations to which people living around the TSDF would most likely be exposed. The results are presented in Table 7. It is important that these estimates be

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[30] California Department of Health Services, California Air Resources Board and South Coast Air Quality Management District. "Ambient Air Monitoring and Health Risk Assessment for Suspect Human Carcinogens around the BKK Landfill in West Covina," 1983.

TABLE 7

PRELIMINARY ESTIMATES OF INCIDENCE AND INDIVIDUAL RISKS ASSOCIATED WITH AIR RELEASES FROM ONE TREATMENT, STORAGE, AND DISPOSAL FACILITY (TSDF)

Pollutants Having Some Evidence of Carcinogenicity*	Concentration**(ug/m^3)		Preliminary Approximation of Individual Lifetime Risk***	
	Max	Min	Max	Min
Benzene	3.8	0.0	$2.6x10^{-5}$	0.0
Chloroform	1.0	0.0	$1.0x10^{-6}$	0.0
Vinyl chloride	12.1	0.0	$3.2x10^{-5}$	0.0
Perchloroethylene	6.8	0.0	$1.2x10^{-5}$	0.0
Trichloroethylene	5.4	2.1	$2.2x10^{-5}$	$8.6x10^{-6}$
Ethylene dichloride	6.3	0.8	$4.4x10^{-5}$	$5.6x10^{-6}$
Total Additive Lifetime Risk			$1.4x10^{-4}$	$1.4x10^{-5}$

* The weight of evidence of carcinogenicity for the compounds listed varies greatly, from very limited to very substantial. Further, the extent of evaluation and health review performed varies considerably among compounds. However, for the purposes of this report, a conservative scenario (i.e., that all compounds examined could be human carcinogens) has been assumed.

** Concentration data source: California Department of Health Services, California Air Resources Board and South Coast Air Quality Management District, "Ambient Air Monitoring and Health Risk Assessment for Suspect Human Carcinogens Around the BKK Landfill in West Covina," 1983.

*** Because of the uncertainties in the data used to make these estimates, they should be regarded as rough approximations of lifetime individual risk. These estimates are drawn from measurements made at one TSDF, and should not be considered representative of usual TSDF emissions, but rather illustrative of potential TSDF emissions.

interpreted as an isolated example, providing only a rough indicator of potential risk from TSDF's. However, the estimates exhibited in this table suggest that risks around this landfill are similar to those near major point sources. The lifetime individual risks for the highest observed values range from 10^{-5} to 10^{-6}, and the maximum additive lifetime individual risk for the six compounds is 1.4×10^{-4}.

Superfund Sites

There is evidence suggesting that uncontrolled or abandoned hazardous waste facilities, e.g., Superfund sites, may be significant sources of air toxic releases. Information provided by the Hazard Ranking System (HRS) [40 CFR Part 300: Appendix A], is one indication of this potential.

For an abandoned hazardous waste site to be listed as a Superfund site and placed on the National Priorities List (NPL), the site must receive a specified score using the HRS. In the HRS, air releases must be both significantly above background concentrations and also "observed" (that is, measured) to receive a score. In contrast, only a "potential" for release to surface or ground water is required in the HRS. The requirement for an observed release for air resulted from a lack of any better method for considering the air route: no good, consistent correlation has been found between physical and chemical properties of wastes and their potential for air migration.

To date, 109 sites have been placed on the NPL due to high air scores. Of these, 43 were listed for particulate, heavy metal, or radium releases. The remaining 67 sites were listed because of volatile organic compound emissions. These 109 facilities represent a total of 16 percent of all currently listed NPL sites.

Municipal Waste Disposal: Incinerators and Landfills

Few attempts have been made to assess the risks that may be attributable to air toxics emissions from municipal incinerators and municipal landfills. Our search for risk assessments on municipal waste treatment led to only one study designed specifically for assessing risks. In this study, dioxin emissions from six municipal incinerators were measured, and maximum individual risks estimated at levels varying from 10^{-5} to 10^{-6}.[31] The investigators concluded that the levels monitored did not present a public health hazard for the residents living in the immediate vicinity.

In another EPA-sponsored analysis, very preliminary estimates were made of emissions of several metals and organic compounds from municipal incinerators. These estimates indicated that maximum individual risks from poorly run facilities may in certain cases exceed those measured in the dioxin risk assessment described above: well-run facilities appear to pose risks approximately 10 to 100 times less than those of poorly run facilities.[32] These latter estimates could be made only by using a variety of assumptions, since no systematic program has been undertaken to monitor stack emissions from municipal incinerators for the purpose of risk analysis.

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[31] Memorandum from Michael Cook (U.S. EPA Office of Solid Waste and Emergency Response) to EPA's Regional Dioxin Coordinators, "TCDD Emissions from Municipal Waste Combustors," December 16, 1983.

[32] Personal communication from David Sussman, U.S. EPA Office of Solid Waste, June 1984.

We were unable to identify any broad-based studies characterizing risks due to air toxics emissions from municipal landfills. However, there is speculation that emissions may in some cases be high due to decomposing plastics, discarded solvents, and mobilization of volatile organics to the atmosphere by methane gas. Two ad hoc studies performed at municipal landfills on Long Island and in the Los Angeles area provide preliminary confirmation of such speculation. At the Long Island landfill, vinyl chloride was detected in the landfill gases at 90 ppm.[33] At the Los Angeles landfill, landfill gas concentrations of vinyl chloride reached 20 to 30 ppm, and ambient levels near the landfill exceeded those found away from the landfill.[34] In addition, stack emissions of vinyl chloride from a gas collection facility at this same Los Angeles landfill exceeded the vinyl chloride NESHAP emission limit (10 ppm) established for other source categories. Since their initial detection, these emissions have been abated. The Los Angeles air pollution control authorities are currently conducting a monitoring program near selected Los Angeles landfills to evaluate the need for air emissions controls.

Drinking Water Treatment Facilities

EPA's Office of Drinking Water and the Office of Policy Analysis are studying air emissions from aeration facilities at drinking water treatment plants. Aeration is used to remove volatile organics from surface water before it is pumped to residential communities for use.

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[33] Personal communication from Marcus Kantz, EPA Region 3, May 1984.

[34] Personal communication from Edward Camarena, South Coast Air Quality Management District, June 1984.

A second issue regarding these facilities concerns potential air emissions of chloroform from chlorination of drinking water supplies. In a monitoring program conducted in Philadelphia by EPA's Integrated Environmental Management Division, the highest ambient concentrations of chloroform found in the city were measured on the grounds of the drinking water treatment plant. However, these findings are still preliminary and must be examined in greater detail.

Sewage Sludge Incineration

EPA's Office of Water Regulations and Standards and the Office of Policy Analysis are examining the issue of air emissions from sewage sludge incineration. The Water Office is specifically interested in whether the New Source Performance Standard (NSPS) for sewage sludge incinerators promulgated under the Clean Air Act is adequate. The NSPS regulates emissions of particulate matter, but does not consider the potential health effects of the toxic constituents of those emissions.

PATHWAYS

Ingestion

This study considers only the effects of inhaling toxic air pollutants. The quantitative risks due to human ingestion of air pollutants are not covered, although there are several examples of the ways that toxic air emissions may be ingested. In Tacoma, Washington, researchers discovered that children living near the ASARCO copper smelter have elevated levels of arsenic in their urine; one possible exposure route is by ingestion of contaminated soil. Fish in Lake Superior contain toxaphene that was deposited in the lake after being carried by the wind from areas where toxaphene was used as a pesticide. In Maryland, some analyses suggest that as much as 30 percent of the metals present in the Baltimore Harbor may have been air-deposited, either by direct deposition from the air or indirectly through urban runoff. Finally, half of the 1,000+ chemicals inventoried in the Great Lakes appear to result at least in part from air deposition.

Stratospheric Ozone Depletion and Skin Cancer

The analysis did not consider the possible health effects caused by a reduction in the stratospheric ozone layer. Carbon tetrachloride, and other chlorinated organics with long atmospheric lifetimes, have the potential to affect the ozone layer, and could indirectly increase the incidence of skin cancer. For example, it is estimated that by the year 2020, U.S. emissions of carbon tetrachloride could be responsible for between 500 and 22,000 excess cases of skin cancer annually in the U.S., resulting in 3-220 excess deaths per year.[35]

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[35] Zaragoza, L. "Calculating jEffects of Carbon Tetrachloride and Other Chlorocarbons on Increases in Skin Cancer from Stratospheric Ozone Depletion," EPA, OAQPS Draft. July 25, 1984.

C. Summary of the Magnitude of the Air Toxics Problem

Estimated risks from air toxics have been presented for each major analytical study: the NESHAP Study, the 35-County Study, and the Ambient Air Quality Study. The results differ among the three studies because of differences in technical approaches, pollutants and sources covered, and emissions estimates, making interpretation and integration of the disparate results difficult. A useful statistic for summarizing the results of all three studies seems to be annual incidence per million population. Table 8 summarizes this statistic for the 17 pollutants and pollutant groups for which sizeable risks were estimated in any of the analyses.

It should be noted that estimates were derived differently in each of the studies: those from ambient air data weighted urban and rural population and concentrations to arrive at a national average; the national aggregate values calculated for the NESHAP Study and for asbestos, radionuclides, and gasoline marketing were spread over the total national population of 230 million; and the population living in the 35 counties was used to calculate incidence per million for the 35-County Study.

The estimated annual incidences per million people for the pollutants included in this report were 5.6 for the NESHAP analysis, 7.4 for the Ambient Air Quality Study, and 4.9 for the 35-County Study. These totals are surprisingly close. However, this closeness is somewhat coincidental and disguises large inconsistencies in the pollutant-by-pollutant estimates. For instance, chromium accounts for only 0.29 cases per million in the 35-County study and 1.43 in the NESHAP analysis. Volatile organic compounds contribute a total of 2.6 per million based on the ambient measurements and only 0.6 for the NESHAP data.

TABLE 8

SUMMARY TABLE: PRELIMINARY APPROXIMATION OF ANNUAL INCIDENCE ESTIMATES PER MILLION POPULATION FROM THE NESHAP STUDY, THE AMBIENT AIR QUALITY STUDY AND THE 35-COUNTY STUDY**

Pollutants Having Some Evidence of Carcinogenicity*	NESHAP Study	Ambient Air Quality Study	35-County Study
Six Month Study Risk Estimates			
Formaldehyde	0.01	0.83	0.21
Benzene	0.14	1.02	0.39
Chromium†	1.43	1.05	0.29
Cadmium	0.04	0.06	0.02
Arsenic	0.02	0.26	0.02
Trichloroethylene	0.04	0.08	0.15
Perchloroethylene	0.01	0.10	0.14
Ethylene oxide	0.21	N/A	N/A
Carbon tetrachloride	0.06	0.19	0.004

* The weight of evidence of carcinogenicity for the compounds listed varies greatly, from very limited to very substantial. Further, the extent of evaluation and health review performed varies considerably among compounds. However, for the purposes of this report, a conservative scenario (i.e., that all compounds examined could be human carcinogens) has been assumed.

** Because of the uncertainties in the data used to make these estimates, they should be regarded as rough approximations of incidence. Estimates for individual compounds are much less certain. These incidence estimates have been performed to provide a rough idea of the possible total magnitude of the air toxics problem, and will be used only for priority-setting and to provide policy guidance.

† Risk estimates assume that all species of chromium are carcinogenic, although only certain species have evidence of carcinogenicity. Current data do not allow differentiation among species.

TABLE 8 (Cont.)

SUMMARY TABLE: PRELIMINARY APPROXIMATION OF ANNUAL INCIDENCE ESTIMATES PER MILLION POPULATION FROM THE NESHAP STUDY, THE AMBIENT AIR QUALITY STUDY AND THE 35-COUNTY STUDY**

Pollutants Having Some Evidence of Carcinogenicity*	NESHAP Study	Ambient Air Quality Study	35-County Study
Ethylene dibromide	0.12	N/A	0.02
Chloroform	< 0.01	0.07	0.002
Vinylidene chloride	< 0.01	0.27	N/A
Gasoline vapors	N/A	N/A	0.15
All other	0.11	0.01	0.38
Risk Estimates from Other EPA Efforts			
Radionuclides	0.07	0.07	0.07
Asbestos	0.50	0.50	0.50
PIC***	2.65	2.65	2.60
Gasoline Marketing	0.20	0.20	
TOTAL	5.6	7.4	4.9

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* The weight of evidence of carcinogenicity for the compounds listed varies greatly, from very limited to very substantial. Further, the extent of evaluation and health review performed varies considerably among compounds. However, for the purposes of this report, a conservative scenario (i.e., that all compounds examined could be human carcinogens) has been assumed.

** Because of the uncertainties in the data used to make these estimates, they should be regarded as rough approximations of incidence. Estimates for individual compounds are much less certain. These incidence estimates have been performed to provide a rough idea of the possible total magnitude of the air toxics problem, and will be used only for priority-setting and to provide policy guidance.

*** "Products of Incomplete Combustion" (PIC) refers to a large number of compounds, probably consisting primarily of polynuclear organics. The PIC unit risk value was derived from dose-response data that use BaP levels as a surrogate for PIC or total air pollution. There are many limitations of using the BaP surrogate method to estimate PIC risks. Thus, all PIC estimates presented in this report must be regarded as highly uncertain. Refer to pp. 20-25 for a more detailed explanation of how the PIC unit risk value was derived.

A major contributor to these estimates is the pollutant category we have labeled products of incomplete combustion (PIC). It is unique among the pollutants examined and deserves special mention. PIC is used in this study to represent a large number of air pollutants associated with lung cancer in epidemiological studies of people exposed to those pollutants in the 1940's and 1950's. We assumed that these exposures were dominated by PIC. The unit risk factor was derived by using BaP as a surrogate for PIC, and is based on these epidemiological studies. This method of quantifying risk is unusual, and the fact that major risks are estimated for PIC makes the calculation controversial. The alternative is to exclude PIC and to ignore the implications of the epidemiological studies and the contribution of these compounds, some of which are proven carcinogens. More detail on the derivation of the unit risk value for PIC is provided on pages 20 to 25.

Although incidence per million population is an important statistic, aggregate national totals also provide perspective and allow comparison with other cancer statistics. The annual incidence estimates derived from the incidence rate for the major analyses (Table 8) are:

NESHAP Study - 1,300 (national estimate)
Ambient Air Quality Study - 1,700 (national estimate)
35-County Study - 230 (for 35 counties only)

Most of our analyses also estimated individual lifetime risk. As opposed to aggregate incidence, which applies to an entire population, individual lifetime risk describes the risk to a specific individual at a specific location (usually the worst-case site). It almost always occurs within 0.1 km and 0.3 km from the fenceline of major sources. The values are very susceptible to errors in modeling assumptions, population location, and emission estimates, and it is difficult to interpret the results of national studies. In our analysis, maximum risks near point sources frequently reached one in a thousand (10^{-3}) or greater and were routinely greater than 10^{-4}. For example, in the NESHAP study, 13 pollutants presented a maximum individual risk of 1×10^{-3} or greater in at least one location, and 21 pollutants (nearly half of those studied in the NESHAP analysis) presented risks greater than 1×10^{-4}.

The ambient air data were used to calculate an aggregate individual risk for multi-pollutant exposures. Since these aggregate individual risks were based on measured data for a specific sampling site, they were subject to less uncertainty than most of the risk estimates in this report and may be used as an important indicator of the general magnitude of the urban air toxics problem. However, the amount of data available falls short of that needed for a comprehensive analysis of any of the urban areas, and the results should not be used for city-to-city comparison.

Since reasonably complete monitoring data were needed to estimate these aggregate risks, only a few urban areas with the best data bases could be included. Generally, these were large cities with medium to heavy industrialization. The additive risks ranged from 0.7×10^{-3} to 2.3×10^{-3}, based on measurements of two to three metals, BaP as an indicator for PIC's, and 6 to 10 volatile organics monitored at the same or very proximate locations (Table 5). These locations generally were in city centers and were not associated with specific point sources.

It is not possible to estimate the number of people exposed to such multi-pollutant risks. However, it is interesting to compare them to the estimates of annual incidence per million reported earlier. A lifetime risk of 2.3×10^{-3} equals 2,300 excess cancer cases per million population for a 70-year period, or 33 per million per year; a lifetime risk of 0.7×10^{-3} equals about 10 per million cancer cases per year.

D. Perspective and Context: Other Cancer Risks

One way to evaluate the importance of the air toxics risks described above is to compare them with risks linked to other factors. For example, Doll and Peto estimate that about 65 percent (286,000) of annual cancer deaths appear to be related to smoking (30 percent) or diet (35 percent), and that about 2 percent of total cancer deaths (8,800) are associated with environmental pollution.[36]

The magnitude of the air toxics problem presented in this study is given for PIC in terms of cancer deaths, and as cancer cases for other pollutants. Therefore, these risks should be compared both to statistics regarding both total cancer cases and cancer deaths. Table 9 presents projected estimates of 1983 cancer mortality and morbidity made by the American Cancer Society (ACS).[37] This table shows that about 850,000 cancer cases and 440,000 cancer deaths were projected for 1983. The ACS reports also that 135,000 lung cancer cases and 117,000 lung cancer deaths were projected for 1983.

If indoor air exposures are considered, this study may not accurately estimate the potential number of cancers associated

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[36] Doll, Richard, and Richard Peto, "The Causes of Cancer: Quantitative Estimates of Avoidable Risks of Cancer in the United States Today," Journal of the National Cancer Institute. June 1981.

[37] American Cancer Society, 1982. Cancer facts and figures, 1983.

TABLE 9

PERSPECTIVE AND CONTEXT: STATISTICS ON CANCER RISKS

TOTAL ESTIMATED CANCER CASES (1983)[1]	850,000	(3,700/million)
TOTAL ESTIMATED CANCER DEATHS (1983)[1]	440,000	(1,900/million)
Diet[2]	154,000	(670/million)
Smoking[2]	132,000	(570/million)
Environmental pollution[2]	8,800	(38/million)
CANCER CASES ASSOCIATED WITH INDOOR AIR EXPOSURES		
Radon[3]	1,000 to 20,000	(4 to 91/million)
Passive smoking[4]	500 to 5,000	(2.2 to 22/million)
Formaldehyde[3]	160	(0.7/million)
Other organic compounds[5]		
Carbon tetrachloride	340	(1.5/million)
Benzene	500	(2.2/million)
Chloroform	240	(1.1/million)
Tetrachloroethylene	200	(0.9/million)
Trichloroethylene	220	(1.0/million)

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[1] Source: American Cancer Society, 1982. Cancer Facts and Figures, 1983.

[2] These estimates are presented for illustrative purposes only, since many consider that such attribution of cancer cases to a particular exposure oversimplifies the multi-causal nature of cancer. The estimates were derived by combining the estimated percent of cancer deaths attributed to diet, smoking, and pollution presented in Doll and Peto (reference 35) with the American Cancer Society estimates of total 1983 cancer deaths.

[3] These estimates were made by the relevant EPA program offices. For specific references and a discussion of these estimates, refer to Thomson, Vivian, "Indoor Air Pollution: Ramifications for Assessing the Magnitude and Nature of the Air Toxics Problem in the United States," U.S. EPA Office of Policy Analysis, September 1984.

[4] Repace, J. L., and A. H. Lowrey, "A Quantitative Estimate of Non-smokers' Lung Cancer Risk from Passive Smoking," EPA, in press.

[5] These values are calculated from personal exposure monitoring data collected in EPA's Total Exposure Assessment Methodology Study (TEAM), which weights them heavily toward the impact of indoor exposures. They are taken from: Wallace, Lance, "Review of Air Toxic Document," Memorandum to Bern Steigerwald, EPA, October 30, 1984.

with exposures to toxic air pollutants. Historically, indoor, nonoccupational air quality has been virtually ignored by EPA and other federal agencies, despite the fact that average Americans spend about 80 to 90 percent of their time indoors. Recent data show that indoor radon exposures may cause from 1,000 to 20,000 lung cancer cases annually, and EPA estimates show that 500 to 5,000 cancer cases may be caused by passive smoking.[38] In addition, indoor levels of formaldehyde routinely exceed outdoor levels by an order of magnitude, while indoor levels of other organics--such as benzene, trichloroethylene, and tetrachloroethylene--may exceed outdoor levels by 2 to 5 times for the median-exposed individual and up to 50 times for the most-exposed individual.[39] Preliminary risk estimates (Table 9) for indoor plus outdoor exposures to five organic compounds greatly exceed those based on ambient levels only (Table 5). Combined with the large amount of time that Americans spend indoors, these data indicate that our estimates of the magnitude of the air toxics problem--based only on outdoor ambient levels--may understate the extent of the air toxics problem for those compounds that can be emitted indoors.

It is also possible that our analysis has somewhat overstated risks due to the metals examined in the study. No indoor versus outdoor data could be found for the specific metals examined in this study. However, there are limited data indicating that other trace metals (e.g., vanadium, manganese) show indoor/outdoor ratios somewhat less than 1.0.[38]

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[38] Thomson, Vivian, "Indoor Air Pollution: Ramifications for Assessing the Magnitude and Nature of the Air Toxics Problem in the United States," U.S. EPA Office of Policy Analysis, September 1984.

[39] Wallace, Lance et al. "Total Exposure Assessment Methodology (TEAM) Study: First Season - Northern New Jersey." Interim Report. U.S. EPA, Office of Research and Development.

Limited data are available characterizing the cancer risks due to ambient environmental exposures other than air pollution. As part of this study, EPA's Chemical Coordination Staff attempted to compare risk levels triggering regulation across several of EPA's program offices. The staff concluded that such comparisons are difficult to make, since EPA has in fact made few regulatory decisions for carcinogens based on quantitative risk assessment. However, a few examples of risk-assessment based decisions were found. For instance, EPA recently banned most uses of the pesticide ethylene dibromide after estimating that EDB exposures might cause as many as 13,000 cancer cases per year. EPA has also banned most uses of chlordane/heptachlor, based on estimates of 500 cancer cases caused annually, and the asbestos school inspection program was started after risks were estimated at approximately 60 cancer cases annually.[40]

As previously discussed, the maximum individual risks estimated in this study ranged widely, from 10^{-1} to less than 10^{-6}. Risks of 10^{-3} and greater were commonly estimated for major point sources, and the combined lifetime individual risks based on ambient data were in the 10^{-3} range. The Chemical Coordination Staff's analysis shows that, on average, EPA has taken regulatory action based on maximum individual risks in the 10^{-3} to 10^{-4} range, although there may be differences among program offices:

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[40] Viviani, Donn et al., "Acceptable Risk Levels and Federal Regulations: A Comparison of National Emission Standards for Hazardous Air Pollutants (NESHAP) with Other Federal Standards Based on Quantitative Risk Assessment," U.S. EPA Office of Pesticides and Toxic Substances, May 1984.

> Although the data are somewhat limited, the Office of Air and Radiation generally appears to use a marginally higher level of individual risk (both before and after regulation) than other offices. However, when viewed from an aggregate risk perspective, risks to the total population are not much different from those of other offices.[39]

V. NATURE OF THE AIR TOXICS PROBLEM

Whereas previous sections of this report focused on the magnitude of the national air toxics problem, the following section will discuss the causes of air toxics exposures and risks. Four questions will be addressed, using the results of the studies and analyses previously discussed:

1. What pollutants appear to cause most of the air toxics problem as we understand it now?
2. What sources appear to be major contributors to air toxics risks?
3. Do air toxics problems vary geographically?
4. Can we estimate the degree to which indirect control of air toxics is affected through the criteria pollutant programs?

A. Pollutants

Table 8 shows that approximately 15 pollutants and pollutant groups account for most of the cancer risks examined in this study: PIC, chromium, benzene, arsenic, cadmium, carbon tetrachloride, chloroform, ethylene dibromide, ethylene oxide, formaldehyde, gasoline vapors, perchloroethylene, trichloroethylene, asbestos, and radionuclides. Thus, it appears that the pollutants responsible for most of the cancer cases associated with air toxics consist of a mixture of metals, volatile organic compounds, and products of incomplete combustion. Many of these same pollutants (for example, chromium, benzene, ethylene oxide, and arsenic) also show maximum individual risks in the 10^{-1} to 10^{-3} range.

An interesting feature of the analysis is the relatively low aggregate risk estimated for many of the synthetic organic chemicals: national incidence totalled less than 1.0 cancer cases per year for 21 such compounds. This fact is noteworthy, since it has been speculated that such "exotic" chemicals may be major sources of air toxics risks. However, these low incidence estimates are based on exposure modeling, and have not been verified by ambient data. In addition, maximum individual risks associated with some of these chemicals ranged up to 10^{-3}.

B. Sources

Not surprisingly, an examination of emissions associated with the pollutants listed above shows a diverse and complex group of sources. Table 10 gives a source breakdown for several of the more important pollutants examined in the study. For example, chromium is emitted from such major point sources as steel and refractory manufacturing facilities, as well as from fuel combustion. Formaldehyde is emitted from mobile sources, chemical plants, fuel combustion, indoor sources (such as particleboard), and is formed photochemically in the atmosphere.

TABLE 10

SOURCES OF SELECTED COMPOUNDS EXAMINED IN THIS STUDY

Pollutant	Sources
Arsenic	Combustion sources such as waste oil burning, utility boilers (coal-fired), wood smoke, smelters, glass manufacturing
Benzene	Road vehicles, gasoline marketing, petroleum refining
Chloroform	Solvent usage, water treatment
Chromium	Waste oil burning, steel manufacturing, refractory manufacturing, metals manufacturing, combustion sources
Ethylene oxide	Chemical industry, sterilant
Formaldehyde	Road vehicles, formaldehyde manufacturing, petroleum refining, oil and gas combustion
Perchloroethylene	Solvent usage, dry cleaning facilities
PIC*	BaP sources include use of wood and coal in small combustion units, coke operations, internal combustion engines
Trichloroethylene	Solvent usage

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* "PIC" is shorthand for Products of Incomplete Combustion, a broad and ill-defined group of compounds represented in this study by BaP, an organic particulate. The mix of compounds present will vary from different combustion processes.

The complexity and diversity of air toxics sources are underscored by the following observations concerning emissions of the most significant pollutants listed in Table 8.[41]

- Manufacturing facilities for synthetic organic chemicals are responsible for greater than 20 percent of total national emissions for only 3 of the major pollutants.
- Mobile sources account for greater than 20 percent of emissions for only 3 of the major pollutants.
- Solvent usage is responsible for greater than 20 percent of emissions for only 3 of the major pollutants.
- Fuel combustion in stationary sources accounts for greater than 20 percent of emissions for only 4 of the major pollutants.

Another perspective on which source types appear to be important contributors to the air toxics problem can be had by using the individual risk or incidence estimates from the NESHAP and the 35-County Studies. For pollutants that were evaluated directly, area and point sources each accounted for about half of the aggregate incidence in both the NESHAP and 35-County Study. When PIC is included (using BaP as a surrogate) area sources become more dominant, accounting for over 75 percent of the incidence in both the 35-County and NESHAP studies. This result is consistent with the fact that PIC is estimated to account for a large portion of aggregate incidence, and that nearly all BaP emissions appear to come from area sources (principally motor vehicles, and combustion of wood, coal, and oil in small heating units). The contribution of the most significant source types based on cancer incidence as determined by the 35-County Study are shown in Table 11.

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[41] Lahre, Tom, "Characterization of Available Nationwide Air Toxics Emissions Data," EPA Contract No. 68-02-3513, Task No. 46, June 1984.

The high proportion of total incidence that apparently is due to road vehicles as shown in Table 11 merits additional discussion. To estimate incidence attributable to PIC, the 35-County Study used emission estimates to model ambient levels of BaP and then applied the PIC unit risk factor to those modeled ambient concentrations. An alternative method recently brought to our attention applied a diesel emission potency that relates expected cancer incidence specifically to diesel particulate emissions.[42,42a] EPA's Office of Mobile Sources has applied this alternative technique and estimated that after implementation of EPA's recent rule on heavy duty diesel particulate emissions, the incidence rate in the year 2000 would be 1-4 per million population for urban areas. By comparison, the 35-County Study estimated a 1982 incidence of 120 for all mobile vehicle emissions (including gasoline engines) for 45 million people or a rate of 2.7 per million.

The second measure of risk used in this study is maximum individual risk. The NESHAP Study indicates that the highest individual risk for a pollutant is generally associated with large point sources.

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[42] "Control of Air Pollution From New New Motor Vehicles and New Motor Vehicle Engines; Gaseous Emission and Particulate Emission Regulations," 40 CFR Parts 86 and 600, Vol. 50, No. 51, Friday, March 15, 1985.

[42a] "Diesel Particulate Study," U.S. Environmental Protection Agency, Office of Mobile Sources, Emission Control Technology Division, October 1983.

TABLE 11

PERCENT OF INCIDENCE ASSOCIATED WITH POINT AND AREA SOURCES BASED ON THE 35-COUNTY STUDY*

Point Sources	% Total Incidence (w/o PIC)	% Total Incidence (w/PIC)
Chemicals Production	11	4
Metals Manufacturing	8	3
Petroleum Refining	5	2
Rubber Production	5	2
Utilities	4	1
POTWs	3	1
All Other	11	4
TOTAL PERCENT: POINT SOURCES	47	15
Area Sources		
Road Vehicles	23	60
Solvent Usage	11	4
Gasoline Marketing	9	3
Waste Oil Burning	9	3
Heating		
Wood smoke (stoves/fireplaces)	0.5	12
All other	1.5	3
TOTAL PERCENT: AREA SOURCES	53	85

* Because of the uncertainties in the incidence estimates used to derive these estimates, they should be regarded as rough indicators only. These computations have been performed to provide a rough idea of the nature of the air toxics problem, and will be used only for priority-setting and to provide policy guidance.

C. Geographic Variability

A final method of characterizing the nature of the air toxics problem is to examine geographic variability in ambient air quality and in cancer risks from air toxics. Mean ambient concentrations for selected metals and organic compounds are shown for several cities in Table 12. These data may be for different years and are not for matched sites; therefore, detailed comparison is not warranted. However, they do indicate that ambient levels of toxic air pollutants can vary widely from city to city, with ratios commonly ranging from 5/1 to 10/1.

Although information is scarce, the data available suggest that intercity variation of risk also may be significant. Even with sparse monitoring networks, limited geographical areas within a city are observed with air quality for many air toxics 3 to 10 times the urban average. Two peer reviewers noted the existence of such air quality variation in metropolitan areas and commented on their possible importance in evaluating and regulating the problem of air toxics.[43,44]

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[43] Ferrand, E.F., Department of Environmental Protection, City of New York, letter to Dr. Terry Yosie, Director EPA Science Advisory Board; December 18, 1984.

[44] Lioy, P.J., New York University Medical Center, letter to Dr. Terry Yosie, EPA; October 23, 1984.

TABLE 12

COMPARISON OF MEASURED AIR QUALITY FOR SELECTED CITIES AND POLLUTANTS

	City						
Pollutant	A	B	C	D	E	F	G
Arsenic*	7.4	3.7	3.2	33.5	7.0	----	6.0
Benzo(a)pyrene*	1.7	0.5	0.2	0.3	0.2	----	0.4
Chromium*	93.5	9.3	25.3	13.4	17.0	----	60.0
Benzene**	11.0				14.8	15.7	9.5
Carbon tetrachloride**	4.2				0.3	2.4	2.6
Chloroform**	9.9				0.4	1.5	7.9
Trichloroethylene**	1.4				2.0	0.4	2.8

* Concentrations expressed in nanograms/m^3.

** Concentrations expressed in micrograms/m^3.

The 35-County Study also allowed us to examine the ways in which risks vary among counties. The results are shown in Table 13 (PIC was excluded from this data set because the uncertainty in the emission estimates for BaP make detailed city-specific comparisons especially unreliable). For example, the percent of risk from point sources varies from 52 percent in County 4 to 25 percent in County 2. Similarly, petroleum refining accounts for 22 percent of total risk in County 2, but 0 percent in Counties 3 and 4. There are, however, source categories (road vehicles and waste oil burning) that account for approximately the same percent of risks across counties, primarily because these risks are strongly linked to population. Thus, two main types of sources appear to emerge from the analysis: sources accounting for approximately equal portions of risk from one area to the next, and sources peculiar to a particular area. While the data bases used in these analyses are inadequate to accurately define most areas' air toxics problems, the data do support the intuitive prediction that reducing cancer risks from air toxics will necessitate dealing with certain types of problems at the local level.

If we consider air toxics emissions data, we also find regional variation. For example, of the 93 compounds covered in the emissions study[45], a large concentration of organic substances were found to be produced in an area stretching from Corpus Christi, Texas to New Orleans, Louisiana. Eighteen organic compounds are produced

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[45] Lahre, Tom, "Characterization of Available Nationwide Air Toxics Emissions Data," EPA Contract No. 68-02-3513, Task No. 46, June 1984.

TABLE 13

COMPARISON OF SOURCES OF RISK IN SEVERAL COUNTIES SELECTED FROM 35-COUNTY STUDY[1,2]

	County 1	County 2	County 3	County 4	County 5	All 35 Counties Combined
Percent of incidence from area sources, point sources, and POTW's						
Area	61	66	48	41	67	51
Point	38	25	50	52	32	46
POTW's	1	9	2	7	1	3
Percent of incidence from given source categories						
Road vehicles	31	26	23	14	31	23
Petroleum refining	13	1	22	0	0	5
Chemical production	5	3	21	24	2	10
Solvent usage	8	18	5	10	17	10
Waste oil burning	8	11	9	12	10	8
Percent of incidence from given pollutants						
Formaldehyde	18	7	29	5	30	12
Chromium	9	14	8	10	12	17
Benzene	30	24	24	20	25	23
Vinyl chloride	2	0	2	25	0	11
Perchloroethylene	10	10	3	6	11	8

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[1] For pollutants evaluated directly; excludes PIC.

[2] Because of the uncertainties in the incidence estimates used to derive these estimates, they should be regarded as rough indicators only. These computations have been performed to provide a rough idea of the nature of the air toxics problem, and will be used only for priority-setting and to provide policy guidance.

entirely in Texas and Louisiana, and almost 50 percent of the remaining organic compounds examined in the emissions study are manufactured in those two states. As noted earlier in the report, emissions of many of the synthetic organics are associated with only very low annual incidence.

D. Indirect Control of Air Toxics

Toxic compounds are emitted into the atmosphere from many sources that are controlled for criteria pollutants (EPA's criteria pollutants are: carbon monoxide, ozone, lead, total suspended particulates, oxides of nitrogen, and sulfur dioxide). Metals and polynuclear compounds usually are emitted as particulate matter and most of the volatile organic compounds are ozone precursors. As such, they are regulated under State Implementation Plans (SIP's), New Source Performance Standard (NSPS) program, and Title II for motor vehicles. Also, emissions of some of the compounds--especially solvents--are accomplished for economic reasons to recover lost product or energy.

In attempting to evaluate available analyses on the effects of such indirect control of toxic air compounds, we found two studies.[46] One focused on nine potential air toxics (including benzene, chloroform, and chromium) and evaluated the impact of existing regulations on major point sources. Control of metals from point sources was generally high, ranging from 80 to 98 percent. Much more variation and less control was apparent for organics, with the percentage control ranging from 30 to 90 percent.

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[46] Lahre, Tom. Op. cit.

A second study was less quantitative but provided estimates for 37 compounds and included area sources and motor vehicles. Air quality trends, rather than control regulations, were evaluated to estimate the indirect control of metallic particulates. Generally, reductions of 30 to 70 percent have been observed since the 1960's. In addition, SIP's and NSPS are credited with reducing emissions of 15 chemicals from the chemical industry by 10 to 80% and 8 solvents by 30% nationwide. Motor vehicle controls remove up to 90% of several potentially toxic compounds from exhaust gases.

A more recent analysis compared air quality and emissions data for 1970 with the estimates of incidence for 1980 provided for this report in the Ambient Air Quality Study analysis.[47] Methods, assumptions, and pollutants included were held constant over the period. The calculations showed a significant decrease in incidence during the decade due to improvements in air quality, presumably related to general regulatory programs. The annual incidence rate for the 16 pollutants studied dropped from 17.5 per million using 1970 data to 6.8 per million in 1980. Estimated nationwide incidence decreased from 3600 in 1970 to 1600 in 1980.

Even from these cursory analyses, it is apparent that indirect control can be very significant for toxic compounds. At this time, it appears that control under criteria pollutant provisions of the Clean Air Act far exceeds the impact of Section 112 regulations. Also, since sources are already controlled by criteria pollutant programs, the remaining risks will probably be more difficult to control.

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[47] Hunt, W. F., Faoro, R. B. and Curran, T. C., "Estimation of Cancer Incidence Cases and Rates for Selected Toxic Air Pollutants Using Ambient Air Pollution Data, 1970 vs. 1980," U.S. EPA. April 1985.

VI. ADEQUACY OF DATA BASES

Quantitatively assessing risks from air toxics exposures poses two principal informational problems. The first involves basic health factors, such as evidence of carcinogenicity, potency, the presence or absence of thresholds, and synergism. These are well-known knowledge gaps basic to cancer risk assessment and strategic discussions on air toxics will not influence their resolution. No attempt was made in this study to use new assumptions or procedures regarding health effects; we relied on techniques and methods in use across EPA.

In the short term, the more relevant problem to understanding the air toxics issue is lack of information on emissions and air quality. These data gaps make it difficult to clearly define problems for many situations and impede policy discussions on risk assessment. The problem is widely recognized and universally frustrating. In the poll of state and local agencies, we interviewed 10 agencies in depth on their air toxics problems. All perceived a need for better emissions data. The contractor who conducted the interviews concluded that "The agencies do not seem to have adequate data that would enable them to perform risk assessments for the toxic pollutants emitted."[48]

With the exception of radonuclides, the study consistently found major weaknesses in the data base for air toxics, both in the coverage and in the quality of information available. If more than one source of data existed, inconsistencies were the norm. Most of the air quality data could not be used for population exposure and were clearly not obtained for risk assessment purposes. Many potentially large source categories could not even be included in the study due to a lack of data. These sources included incineration, hazardous waste disposal, atmospheric formation, and Superfund sites.

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[48] Radian Corp., "Definition of the Air Toxics Problem at the State/Local Level," EPA Contract No. 68-02-3513; Work Assignment 45, June 1984.

Today, air quality data are generally collected to determine trends for criteria pollutants; very few data are available for developing population exposure estimates for toxic air pollutants. Despite significant efforts to assemble monitoring data for all sources, this analysis could only cover about 18 pollutants. Several observations regarding the air quality data are as follows:

- More air quality data were found for metals than for BaP or volatile organics. However, while 170 counties with a total population of about 60 million had monitoring data, only 30 counties had data for more than one site, and essentially no measurements were optimal for exposure assessment.
- Data for BaP were found for about 50 counties. However, most of the measurements were taken three to five years ago and only two areas had data for more than one site.
- For volatile organic compounds, EPA's Office of Air Quality Planning and Standards evaluated over 250 references with thousands of entries for over 40 pollutants. However, even with the most relaxed criteria for data completeness, only five cities had data that allowed estimates of annual averages for more than one site, and two of those five had data only because of the monitoring programs conducted as part of multi-media studies by EPA's Integrated Environmental Management Division.

  EPA does not routinely measure ambient levels of potentially toxic VOCs, and only a few states--e.g., California--routinely gather such data. Of the available reports examined for this analysis, most involve spot measurements for 24 hours or less as part of a special project. Only 45 areas in the nation had one valid calendar quarter's worth of data for any toxic VOC, and only 12 areas had two valid quarters of 5 days each.

Emission inventories for toxic compounds also have major problems. About 250 references were evaluated in this study. Based on this analysis, the most significant concerns were:[49]

- inconsistent coverage of sources;
- highly variable emission estimates;
- poorly defined source categories;
- obvious anomalies and gaps;
- form of metals not shown (speciation);
- poor coverage of dispersive end uses, e.g., solvents; and
- changing data base with time.

To quantify the quality of the emission data available, the reviewers assigned a Confidence Score to each of the 93 pollutants. This subjective rating system is commonly used in evaluating emission inventories. The reviewers' scores are summarized below.

- 5 pollutants, "A" (consistent among information sources; recent detailed study);
- 22, "B" (reasonable agreement among several information sources);
- 59, "C" (sketchy data or significant variability in the estimates);
- 7, "D" (virtually no information found).

The detailed report on emissions also discusses some examples of inconsistencies found in the data. For example, five references were found for chloroform with emissions ranging from 3,999 kkg/year to 11,800 kkg/year (kkg = 1,000 kilograms). For chloroform, the subcategory of solvent use accounted for percentages of total emissions ranging from 6.2 to 92 percent in the various studies and production emissions varied from 1.7 to 11.7 percent. Water chlorination was mentioned as a source of chloroform emissions in only one study.

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[49] Lahre, Tom, "Characterization of Available Nationwide Air Toxics Emissions Data," EPA Contract No. 68-02-3513, Task No. 46, June 1984.

Not only are emissions data scarce and often inconsistent, but systems and institutions are not in place to collect, store, or retrieve data that may become available. There is an almost complete lack of standardization, definition, and data systems. If data are collected, they are collected for a single, short-term purpose.

For monitoring programs, there are no standard methods or guidance available on network design, siting of monitors, and averaging times. The Aerometric Information Retrieval System is being developed by EPA, but until it becomes available in 1987, there is no central repository for air toxics monitoring data.

A comparison with criteria pollutants helps explain why the data base for toxics is relatively inadequate. There are eight pollutants or pollutant categories tracked or regulated under SIPs, while toxic compounds of interest number from 50 to 100. About $30 million per year of EPA grants to state and local agencies are used for gathering data on criteria pollutants, while only about $1 million is used for air toxics. In addition, ambient concentrations of toxics are almost always 100 times less than those of the criteria pollutants. Metals, such as chromium and cadmium, are rarely seen at 0.01 ug/m^3, whereas TSP is measured in tens of ug/m^3. The TSP primary annual ambient standard is set at 75 ug/m^3. Regulation of criteria pollutants is based simply on attainment of a uniform ambient level everywhere. However, toxics regulation often is driven by risk analysis, which requires population exposure estimates and, therefore, a more comprehensive data base. Institutional support has been developed for criteria pollutants over a period of two decades. This infrastructure includes data systems for ambient and emissions data, regulations requiring monitoring networks and comprehensive emission inventories, standard methods of sampling and analysis, and formal quality assurance programs. None of these are yet available for air toxics.

VII. CONCLUSIONS

Given that this analysis was a scoping effort undertaken for purposes of orientation and not to directly support regulation, and considering the omissions and uncertainties discussed in this report, the Study Team believes that the following conclusions can be drawn from this study:

1. Both point sources (major industrial sources) and area sources (smaller sources that may be widespread across a given area, such as solvent usage and motor vehicles) appear to contribute significantly to the air toxics problem. Large point sources are associated with many high individual risks, while area sources appear to be responsible for the majority of aggregate cancer incidence.

2. While there is considerable uncertainty associated with the risk estimates for some substances, available data indicated that the following pollutants may be important contributors to aggregate cancer incidence from air toxics: metals, especially chromium and arsenic; asbestos; products of incomplete combustion; formaldehyde; benzene; ethylene oxide; gasoline vapors; and chlorinated organic compounds such as chloroform, carbon tetrachloride, perchloroethylene, and trichloroethylene; and vinylidene chloride.

3. A wide variety of sources contributes to individual risk and aggregate incidence from air toxics. These include: road vehicles; combustion of coal and oil; woodstoves; metallurgical industries; chemical production and manufacturing; gasoline marketing; solvent usage; and waste oil disposal. As a broad category of activities, combustion/incineration is probably the largest single source of risk.

4. For those cities with sufficient data for analysis, large city-to-city and neighborhood-to-neighborhood variation in pollutant levels and sources was found. However, our current air toxics data base is inadequate to accurately characterize most local air toxics problems.

5. Three analyses quantified estimated cancer risks due to 15 to 45 toxic air pollutants (the number of pollutants

examined varied with the different analyses). The estimates from these analyses showed a range of 5 to 7.4 cases of cancer per million people per year (1,300 to 1,700 cases annually nationwide) for the pollutants examined. These are not actual predictions of incidence, but are instead a statistical way to represent the aggregate risks of pollutants and sources.

The reader is reminded that these estimates are highly uncertain, and is cautioned that the convergence of the various analyses on a seemingly narrow range (5 to 7.5 cases per million) is somewhat coincidental, given that estimates for individual compounds varied widely among the different analyses.

For perspective, estimated nationwide cancer cases and cancer deaths for 1983 were 850,000 and 440,000, respectively.

6. Maximum lifetime individual risks of 10^{-4} (1 in 10,000) or greater in the vicinity of major point sources were estimated for 21 pollutants, about half of those that were studied. Maximum lifetime individual risks of 10^{-3} (1 in 1,000) or greater were estimated for 13 pollutants.

7. Additive lifetime individual risks in urban areas due to simultaneous exposure to 10 to 15 pollutants ranged from 10^{-3} to 10^{-4}. These risks, which were calculated from monitoring data, did not appear to be related to specific point sources. Instead, they represent a portion of the total risks associated with the complex pollutant mixtures typical of urban ambient air.

8. Some low-production organic chemicals appeared to contribute little to aggregate incidence: 21 organic chemicals were estimated to account for a total of less than 1.0 cancer cases per year nationwide. However, this conclusion may be due in part to the lack of data concerning the emissions and toxicity of these "exotic" chemicals.

   Some of these low-production compounds did appear to be associated with high individual risks. For example, the maximum lifetime individual risk for 4,4,-methylene dianiline was estimated at 1.5×10^{-3}.

9. The study indicated that "non-traditional" sources of air toxics--such as publicly owned treatment works (POTW's) and hazardous waste treatment, storage and disposal

facilities (TSDF's)--may pose important risks in some locations. For instance, preliminary findings suggest that POTW's with industrial indirect discharges may emit volatile organic compounds in excess of 100 kkg/yr. Individual lifetime risks for a single compound at one TSDF were estimated as high as 10^{-5}.

10. EPA's criteria pollutant programs appear to have done more to reduce air toxics levels than have regulatory actions aimed at specific toxic compounds. An analysis of 16 pollutants was made using both monitoring and emission data in order to evaluate progress between 1970 and 1980. The estimated cancer incidence rate for these air pollutants in 1980 was less than half that for 1970: 6.8 per million per year in 1980, compared to 17.5 per million in 1970. This seems reasonable considering the diverse array of air toxics sources, the multi-pollutant nature of the problem, and the relative intensity of EPA's criteria and air toxics programs.

11. Even after regulations are implemented under Section 112 of the Clean Air Act for benzene and arsenic, these pollutants still appear to make significant contributions to aggregate incidence due to air toxics. This seems to demonstrate that the base for the air toxics regulatory programs needs to be broadened to include emissions from small area sources, such as combustion, road vehicles, and solvent use.

12. Major weaknesses and gaps characterize air toxics data bases at the federal, state, and local levels. The few air toxics emission inventories available generally show inconsistencies and anomalies, the air quality data available are often inadequate to develop population exposure estimates, and few compounds have been tested adequately for health effects. The data limitations preclude performing specific comprehensive risk assessments for most urban areas, for many compounds, and for many potentially large sources of air toxics risks (such as incineration, hazardous waste disposal, indoor exposures, atmospheric transformation, and Superfund sites).

Sources of Uncertainty

Many assumptions and extrapolaticns are necessary to transform ambient or modeled levels of air pollutants into exposure estimates.

Whether such assumptions introduce a high or low bias into the results is difficult to assess. However, it is clear that the use of such assumptions injects a considerable degree of uncertainty into the analyses.

Some of the factors which may have led the analyses to understate the risk of cancer related to air toxics are as follows:

1. Urban ambient air is characterized by the presence of dozens, perhaps hundreds of substances. Risk estimates for most of these could not be calculated due to data limitations.

2. Indoor concentrations of certain pollutants (e.g., radon, tobacco smoke, formaldehyde, and other volatile organic compounds) are commonly several times higher than outdoor concentrations. The estimated cancer incidences associated with indoor exposures to passive smoking (5,000 annually) and radon (1,000 to 20,000 annually), and 24-hour personal exposures to six organic compounds (1,700 annually) indicate that indoor sources make an important contribution to air toxics risks.

3. Risks due to compounds formed in the atmosphere could not be quantified in the analyses using exposure models, but there are indications that these risks may be significant. For example, formaldehyde is formed in the atmosphere by the breakdown of other organic compounds, and some compounds (e.g., toluene) may be converted into toxic substances through photochemical reactions.

4. Although it has been shown that certain combinations of exposures may have synergistic effects (for instance, smoking and asbestos exposure), all risks were assumed to be additive.

Factors which may have caused the analysis to overestimate cancer risks associated with air toxics are as follows:

1. Cancer unit risk values were obtained from EPA's Carcinogen Assessment Group (CAG) and Clement Associates. EPA unit risk values are generally regarded as plausible, upper-bound estimates. That is, the unit risks are not likely to be higher, but could be considerably lower. In many cases, the unit risk values are preliminary.

2. The weight of evidence of carcinogenicity for the compounds examined varies greatly, from very limited to very substantial. Further, the extent of evaluation and health review performed varies considerably among compounds. For this report, a conservative scenario (i.e., that all compounds included in the report are human carcinogens) was assumed.

3. The risk assessments assume that people living in an area are exposed to the estimated ambient levels for 70 years, 24 hours a day. This especially compromises estimates of maximum lifetime individual risk. Few plants operate for 70 years, most people change their homes several times during their lives, and leave their neighborhoods during the day.

4. The degree to which outdoor emissions of many pollutants (e.g., trace metals) penetrate indoors is largely unknown. If emissions of a pollutant from outdoor sources do not penetrate completely indoors and if there are no indoor sources of that pollutant, then we will have over-stated risks, since we have assumed constant exposure to levels equalling those of outdoor air.

5. Although certain combinations of exposures may have antagonistic effects, all risks were assumed to be additive.

VIII. CURRENT ACTIVITIES

The study was completed during the summer of 1984 and a draft report was circulated for peer review to approximately 20 experts in the field in October 1984. Their technical suggestions are reflected in this final version of the report. Many of the reviewers recommended a final section that would provide information on EPA's response to the principal findings of this analysis. This section responds to that suggestion.

Based on the conclusions in the draft report, EPA initiated a series of intense activities to reexamine its program for air toxics and to evaluate alternative national strategies. Some of these activities include:

- Formation of an Agency-wide Air Toxics Group to guide the review of the study and to assist in the development and implementation of changes to the national program
- Initiation of several additional analyses of the air toxics problem to study 1) the controllability of the most important toxic pollutants, including cost of control to various levels and impact on cancer incidence in several representative cities; 2) the effectiveness of current programs for criteria pollutants and air toxics in reducing risk in several representative cities over the next decade; 3) the existence, intensity, and controllability of high risk areas in several cities caused by concentrations of sources; and 4) an analysis of the feasibility of improving data on emissions and air quality for major urban areas. These studies are scheduled for completion in May 1985.
- Discussion of the results of the study and of possible strategic implications in over 20 presentations to groups representing industry, environmentalists, State and local governments, Congressional staff, and professional organizations.

- Review of the report by groups within EPA who are responsible for implementing programs related to air toxics, including monitoring, emission inventories, methods development, and regulatory analyses; program changes are being made as appropriate;
- Giving increased priority is being given to a pilot program to evaluate and possibly regulate large point sources through a cooperative effort of Federal, State, and local agencies. Acrylonitrile is the pollutant selected and it is typical of many pollutants with aggregate incidence too small to justify national regulation but with high individual risks in the vicinity of some plants.

Although most of the ongoing analyses and discussions will not be completed until mid-1985, it already appears that significant changes to the current national strategy will be recommended to respond to some of the findings in this report. These include shifting the focus of the direct Federal regulatory program from point sources to more complex situations, including area sources that may be responsible for high aggregate incidence. In addition, two new programs are being evaluated: a formal partnership with State and local agencies on regulation of large point sources to provide better coverage for areas of high individual risk; and an initiative directed at larger geographic areas of high risk resulting from the interaction of many sources either in an urban area or in an isolated industrial region.

ATTACHMENT A

SUMMARY TABLE

POLLUTANTS EXAMINED, UPPER-BOUND RISK VALUES, PRELIMINARY APPROXIMATIONS OF INCIDENCE AND MAXIMUM LIFETIME RISK

POLLUTANTS EXAMINED, UPPER-BOUND RISK VALUES, PRELIMINARY APPROXIMATIONS OF INCIDENCE AND MAXIMUM LIFETIME RISK

Pollutants Having Some Evidence of Carcinogenicity*	Unit Risk 1/		Preliminary Approximation of Annual Incidence**				Preliminary Approximation of Incidence Per 10^6 Population**			Preliminary Approximation of Maximum Lifetime Individual Risk** ($x10^4$)	
	Value	Source	NESHAP	35 County 2/	Air Quality	Other	35 County	Air Quality	NESHAP	NESHAP	Air Quality
Acrylamide	1.7x10-5	CLEM	0.01						<0.01	0.74	
Acrylonitrile	6.8x10-5	CAG	0.42	4.2					0.002	38	
Allyl Chloride	5.5x10-8	CAG	<0.01						<0.01	0.01	
Arsenic	4.3x10-3	CAG	4.7	1.1	60		0.02	0.26	0.02	65	40
Asbestos	3/	CLEM					0.5	0.5	0.5		
Benzene	6.9x10-6	CAG	32.3	18.5	234		0.39	1.02	0.14	80	1.5
Benzo-a-Pyrene	3.3x10-3	CAG		1.1	5.4		0.02	0.02			0.25
Benzyl Chloride	1.2x10-5	CLEM	<0.01						<0.01	0.3	
Beryllium	4.0x10-4	CAG	1.2	0.01	0.1		<0.001	<0.001	0.01	1.0	0.002
1,3 Butadiene	4.6x10-7	CLEM	0.01	0.01			<0.001		<0.01	0.1	
Cadmium	2.3x10-3	CAG	8.5	1.1	14.6		0.02	0.06	0.04	36	14.7

* The weight of evidence of carcinogenicity for the compounds listed varies greatly, from very limited to very substantial. Further, the extent of evaluation and health review performed varies considerably among compounds. However, for the purposes of this report, a conservative scenario (i.e., that all compounds examined could be human carcinogens) has been assumed.

** Because of the uncertainties in the data used to make these estimates, they should be regarded as rough approximations of total incidence and maximum lifetime individual risk. Estimates for individual compounds are very uncertain. These incidence and maximum risk estimates have been performed to provide a rough idea of the possible total magnitude of the air toxics problem, and will be used only for priority-setting and to provide policy guidance.

POLLUTANTS EXAMINED, UPPER-BOUND RISK VALUES, PRELIMINARY APPROXIMATIONS OF INCIDENCE AND MAXIMUM LIFETIME RISK

Pollutants Having Some Evidence of Carcinogenicity*	Unit Risk 1/		Preliminary Approximation of Annual Incidence**				Preliminary Approximation of Incidence Per 10^6 Population**			Preliminary Approximation of Maximum Lifetime Individual Risk** ($\times10^4$)	
	Value	Source	NESHAP	35 County 2/	Air Quality	Other	35 County	Air Quality	NESHAP	NESHAP	Air Quality
Carbon Tetrachloride	1.5×10^{-5}	CAG	14	0.2	43		0.004	0.19	0.06	5.8	1.54
Chloroform	1.0×10^{-5}	CAG	0.27	0.1	17		0.003	0.07	<0.01	30	0.77
Chromium†	1.2×10^{-2}	CAG	330.0	13.4	242		0.29	1.05	1.43	1600	14.4
Coke Oven Emissions	6.2×10^{-4}	CAG	8.6	2.4			0.05		0.04	200	
Diethanolamine	1.1×10^{-7}	CLEM	<0.01						<0.01	<0.01	
Dimethylnitrosamine	5.4×10^{-3}	CAG	0.05						<0.01	0.54	
Dioctyl Phthalate	1.3×10^{-7}	CLEM	<0.01						<0.01	0.1	
Epichlorohydrin	2.2×10^{-7}	CAG	<0.01						<0.01	0.02	
Ethyl Acrylate	5.0×10^{-7}	CLEM	<0.01						<0.01	0.47	

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† Risk estimates assume that all species of chromium are carcinogenic, although only certain species have evidence of carcinogenicity. Current data do not allow differentiation among species.

POLLUTANTS EXAMINED, UPPER-BOUND RISK VALUES, PRELIMINARY APPROXIMATIONS OF INCIDENCE AND MAXIMUM LIFETIME RISK

Pollutants Having Some Evidence of Carcinogenicity*	Unit Risk 1/		Preliminary Approximation of Annual Incidence**				Preliminary Approximation of Incidence Per 10^6 Population**			Preliminary Approximation of Maximum Lifetime Individual Risk** ($\times10^4$)	
	Value	Source	NESHAP	35 County 2/	Air Quality	Other	35 County	Air Quality	NESHAP	NESHAP	Air Quality
Ethylene	2.7×10^{-6}	CLEM	<0.01						<0.01	4.9	0.73
Ethylene Dibromide	5.1×10^{-4}	CAG	26.7	1.0			0.02		0.12	1.6	
Ethylene Dichloride	2.6×10^{-5}	CAG	0.9	1.5	11.0		0.03	0.05	<0.01	36	
Ethylene Oxide	3.6×10^{-4}	CAG	47.8						0.21	68	
Formaldehyde	6.1×10^{-6}	CAG	1.6	10.0	191.3		0.21	0.83	0.01	6.1	0.49
Gasoline Vapors	7.5×10^{-7}	CAG		6.8			0.15				
Gasoline Marketing	7.5×10^{-7}	CAG				43					
4,4 ISO Propylidene Diphenol	1.4×10^{-6}	CLEM	0.03						<0.01	<0.01	
Melamine	4.1×10^{-7}	CLEM	<0.01						<0.01	<0.01	
Methyl Chloride	1.4×10^{-7}	CLEM	<0.01		0.9			<0.01	<0.01	0.12	<0.01

* The weight of evidence of carcinogenicity for the compounds listed varies greatly, from very limited to very substantial. Further, the extent of evaluation and health review performed varies considerably among compounds. However, for the purposes of this report, a conservative scenario (i.e., that all compounds examined could be human carcinogens) has been assumed.

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POLLUTANTS EXAMINED, UPPER-BOUND RISK VALUES, PRELIMINARY APPROXIMATIONS OF INCIDENCE AND MAXIMUM LIFETIME RISK

Pollutants Having Some Evidence of Carcinogenicity*	Unit Risk 1/		Preliminary Approximation of Annual Incidence**				Preliminary Approximation of Incidence Per 10^6 Population**			Preliminary Approximation of Maximum Lifetime Individual Risk** ($x10^4$)	
	Value	Source	NESHAP	35 County 2/	Air Quality	Other	35 County	Air Quality	NESHAP	NESHAP	Air Quality
Methylene Chloride	$1.8x10^{-7}$	CAG	1.0		7.4			0.03	0.004	0.1	<0.01
4,4 Methylene Dianiline	$2.1x10^{-5}$	CLEM	0.02						<0.01	15.0	
Nickel (Subsulfide)	$3.3x10^{-4}$	CAG	0.02						<0.01	0.8	
Nitrobenzene	$1.2x10^{-7}$	CLEM	<0.01						<0.01	<0.01	
Nitrosomorpholine	$2.5x10^{-5}$	CLEM	<0.01						<0.01	<0.01	
Pentachlorphenol	$3.9x10^{-7}$	CLEM	0.12	<0.01					0.001	0.17	
Perchloroethylene	$1.7x10^{-6}$	CAG	2.9	6.7	22		0.14	0.10	0.01	4.6	0.19
Products Incomplete Combustion	$0.42x10^{0}$	4/		124	610		2.6	2.65	2.65		31.5
PCBs	$1.2x10^{-3}$	CLEM	0.21						0.001	3.0	

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** Because of the uncertainties in the data used to make these estimates, they should be regarded as rough approximations of total incidence and maximum lifetime individual risk. Estimates for individual compounds are very uncertain. These incidence and maximum risk estimates have been performed to provide a rough idea of the possible total magnitude of the air toxics problem, and will be used only for priority-setting and to provide policy guidance.

POLLUTANTS EXAMINED, UPPER-BOUND RISK VALUES, PRELIMINARY APPROXIMATIONS OF INCIDENCE AND MAXIMUM LIFETIME RISK

Pollutants Having Some Evidence of Carcinogenicity*	Unit Risk 1/		Preliminary Approximation of Annual Incidence**				Preliminary Approximation of Incidence Per 10^6 Population**			Preliminary Approximation of Maximum Lifetime Individual Risk** ($x10^4$)	
	Value	Source	NESHAP	35 County 2/	Air Quality	Other	35 County	Air Quality	NESHAP	NESHAP	Air Quality
Propylene Dichloride	$7.2x10^{-7}$	CLEM	<0.01						<0.01	0.02	
Propylene Oxide	$1.2x10^{-4}$	CLEM	0.97						0.004	300	
Radionuclides	varies	5/				16					
Styrene	$2.9x10^{-7}$	CLEM	<0.01	0.02			<0.01		<0.01	0.33	0.07
Terephthalic Acid	$1.8x10^{-8}$	CLEM	<0.01						<0.01	<0.01	
Titanium Dioxide	$5.6x10^{-7}$	CLEM	0.01						<0.01	<0.01	
Trichloroethylene	$4.1x10^{-6}$	CAG	9.7	6.8	18		0.15	0.08	0.04	1.0	0.26
Vinyl Chloride	$2.6x10^{-6}$	CAG	11.7	8.2			0.2		0.05	38	
Vinylidene Chloride	$4.2x10^{-5}$	CAG	0.04		62			0.27	<0.01	42	0.8

* The weight of evidence of carcinogenicity for the compounds listed varies greatly, from very limited to very substantial. Further, the extent of evaluation and health review performed varies considerably among compounds. However, for the purposes of this report, a conservative scenario (i.e., that all compounds examined could be human carcinogens) has been assumed.

** Because of the uncertainties in the data used to make these estimates, they should be regarded as rough approximations of total incidence and maximum lifetime individual risk. Estimates for individual compounds are very uncertain. These incidence and maximum risk estimates have been performed to provide a rough idea of the possible total magnitude of the air toxics problem, and will be used only for priority-setting and to provide policy guidance.

FOOTNOTES - ATTACHMENT A, SUMMARY TABLE

"Pollutants Examined, Upper-Bound Risk Values, Preliminary Approximations of Incidence and Maximum Lifetime Risk"

1/ The unit risk value is the estimated probability of contracting cancer as the result of a constant exposure over 70 years to an ambient concentration of one microgram per cubic meter (ug/m^3). "CAG" denotes risk values obtained from EPA's Carcinogen Assessment Group; "CLEM" denotes risk values obtained from Clement Associates.

2/ The population of the counties covered in the 35 County Study (about 47.3 million) represents approximately 20% of the national population.

3/ The unit risk value used for asbestos was that a lifetime risk of 10^{-6} for lung cancer would result from an exposure to 10 fibers/cc and that a lifetime risk of 10^{-6} for mesothelioma would result from an exposure to 5 fibers/cc; 30 fibers per nanogram were assumed.

4/ "Products of Incomplete Combustion" (PIC) refers to a large number of compounds, probably consisting primarily of polynuclear organics. The PIC unit risk value was derived from dose-response data which use Benzo(a)Pyrene (BaP) levels as a surrogate for PIC or total air pollution. There are many limitations of using the B(a)P surrogate method to estimate PIC risks: all PIC estimates presented in this report must be regarded as highly uncertain. Refer to pp. 20-25 for a more detailed explanation of how the PIC unit risk value was derived.

5/ Estimates of cancer and genetic risks are based on those found in the 1980 National Academy of Science Report, "Effects on Population of Exposures to Low Levels of Ionizing Radiation" (BEIR - 3 reports).